PB80-177744

Effects of Pollution on Freshwater Fish

(U.S.) Environmental Research Lab.-Duluth, MN

1979

U.S. Department of Commerce
National Technical Information Service
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1. REPORT NO.	2.	3. REC
EPA-600/J-79-073	JOURNAL ARTICLE	PB80-177744

4. TITLE AND SUBTITLE
Effects of Pollution on Freshwater Fish

5. REPORT DATE

6. PERFORMING ORGANIZATION CODE

7. AUTHOR(S)
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8. PERFORMING ORGANIZATION REPORT NO.

9. PERFORMING ORGANIZATION NAME AND ADDRESS
U.S. Environmental Protection Agency
Environmental Research Laboratory-Duluth
6201 Congdon Boulevard
Duluth, Minnesota 55804

10. PROGRAM ELEMENT NO.
1BA820

11. CONTRACT/GRANT NO.

12. SPONSORING AGENCY NAME AND ADDRESS
Same as above

13. TYPE OF REPORT AND PERIOD COVERED

14. SPONSORING AGENCY CODE
EPA-600/03

15. SUPPLEMENTARY NOTES
PUBLISHED: J. Water Pollut. Control Fed. 51(6): 1616-1694 (1979)

16. ABSTRACT

An extensive review of 478 references on the effects of pollution on freshwater fish for 1977-1978 is presented. These references cover: Reviews and Symposia, Dissolved Gases, pH, Insecticides, Herbicides, Piscicides and Anesthetics, Inorganics and Organic Chemicals, Chlorine and Sewage, Pulp and Paper, Radioactivity and Physical Factors

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NATIONAL TECHNICAL
INFORMATION SERVICE
U.S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA. 22161

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
Pollution Freshwater fishes	Toxicity Accumulation Monitoring Aquatic	06/C,F,T

18. DISTRIBUTION STATEMENT	19. SECURITY CLASS *(This Report)*	21. NO. OF PAGES
RELEASE TO PUBLIC	UNCLASSIFIED	79
	20. SECURITY CLASS *(This page)*	22. PRICE
	UNCLASSIFIED	

EPA-600/J-79-073
JOURNAL ARTICLE

Effects of pollution on freshwater fish

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REVIEWS AND SYMPOSIA

Williams *et al.*[1] summarized data collected by the National Eutrophication Survey (NES) to determine the role of nutrients and their sources in 418 eastern lakes and reservoirs. Comparisons between historical and field data of lake conditions and physical, chemical, and biological values obtained from the NES samples indicated that fish kills in several lakes were not related to nutrient concentrations or to water quality parameters such as low dissolved oxygen (DO) concentrations. Entz[2] discussed some of the factors that affected percid fish populations in European waters. Chemical fertilizers and wastewater disposal appeared to have the greatest adverse effects on percid communities. Baily and Denny[3] provided a brief review of past developments that occurred in the Nyumba ya Mungu reservoir (Tanzania) since impoundment. An initial phase of high fish production has been replaced by a slower stabilization phase. It was recommended that new fish should not be introduced into the reservoir to fill gaps in the trophic structure of the fish community.

A book entitled "Wastewater Renovation and Reuse" was edited by D'Itri[4] and included papers dealing with the aquaculture of fish using wastewater and the utility of using fish as indicator organisms for detecting environmental carcinogens. Research needs and public attitudes toward reuse of wastewaters were discussed. Brungs[5] summarized recent studies dealing with the effects of chlorinated condenser wastes on aquatic life and discussed some areas where more research is needed to evaluate the effects of chlorination. Proceedings of a symposium of the European Inland Fisheries Advisory Commission (EIFAC)[6] held in Helsinki, Finland in 1976, was edited by Alabaster. Papers that were included dealt with the classification of river water quality, studies and case histories of fish and invertebrates exposed to pollution, methodology in relation to plankton, and lake monitoring and fisheries.

A bibliography containing 1 695 references on Pacific salmon (*Oncorhynchus* sp.) was prepared by Alderdice *et al.*[7] The subject material dealt with physical, chemical, and biological factors that influence egg deposition and the development and survival of eggs and alevins of this species. Shea and Mathers[8] prepared a bibliography that summarized the physical, chemical, and biological effects of roads on aquatic systems. Many of these papers addressed the effects of silt and turbidity on both fish and fish habitat. Marzolf[9] reviewed the literature pertinent to stream ecology and assessed the impact of clearing and snagging on streams. Clearing and snagging were defined as removal of obstructions from stream banks and channels to increase the streams' capacity to convey water. It was brought out that fish populations would be reduced through removal of cover, shelter, and spawning areas. Alterations of the stream channel also would disrupt territorial behavioral patterns. Changes in walleye (*Stizostedion vitreum vitreum*) populations in the Great Lakes from 1800 to 1975 were reviewed by Schneider and Leach,[10] who linked declining spawning stocks to population, overexploitation, interactions with exotic fish, and degradation of spawning habitat due to logging, damming, increased siltation, and nutrient loading. Of the 21 walleye stocks studied, relatively stable stocks persisted in only five areas, where pollution problems were relatively minor and exploitation light. In addition, these stocks were better able to withstand stresses exerted by alien species. Baxter[11] examined 175 papers on the subject of the environmental effects of dams and impoundments. Part of this review covered the beneficial and detrimental aspects of impoundments, including downstream effects, on fish and fish habitat. Karr and Schlosser[12] reviewed the literature from several research fields to evaluate the effects of near-stream vegetation and channel morphology on water quality and stream biota in agricultural watersheds. A critical review of the current literature on the effects of suspended and dissolved solids on freshwater biota was prepared by Sorenson *et al.*[13] The authors noted that although fishes adapt somewhat to gross changes in salinity, life cycle effects may prevent specific fish from being maintained in a specific aquatic habitat. Relatively high concentrations of suspended solids were needed to cause behavioral reactions (20 000 mg/l) or death (200 000 mg/l) in a short time in fish. Weitkamp and Katz[14] reviewed the world literature regarding the effects of supersaturated dissolved gases with emphasis on gas bubble disease in fish. According to their survey, gas bubble disease was related to total dissolved atmospheric gas levels and not to nitrogen alone. The tolerance of fish to supersaturation was not the same at all life stages and water temperature appeared to have little direct influence on the tolerance of fish to saturation. Critical levels and external signs of the gas bubble disease syndrome were also presented. A brief review paper concerning the relation between acid water and the biology of freshwater fish was prepared by Jacobsen.[15] Such subjects as the effects of acclimation to low pH, tolerance to low pH at different seasons, effects of water temperature on pH tolerance and physiological effects were discussed.

Brungs *et al.*[16] reviewed 338 references concerning the effects of pollution on freshwater fish. Subject matter included water quality, physical and chemical pollutants, and domestic and industrial wastes. A paper that reviewed fish toxicology as a science and as a tool for solving pollution problems was written by Grande.[17] Various types of bioassays (acute, chronic, and screening) were discussed and evaluated. Tubb[18] compiled 10 papers given at a symposium held in Corvallis, Oreg., on recent findings in fish toxicology. Most of the papers presented dealt with the effects of toxic chemicals (kepone, chlorine, cyanide, nitrite, copper, and mixtures of toxic materials) on fish. The remaining papers covered an approach to solving the gas supersaturation problem, collagen metabolism in fish exposed to organic chemicals and an assessment of application factors in aquatic toxicology. Data on 56 life-cycle (chronic) toxicity tests with four species of fish, fathead minnow (*Pimephales promelas*), bluegills (*Lepomis macrochirus*), brook trout (*Salvelinus fontinalis*), and flagfish (*Jordanella floridae*), were examined by McKim.[19] These data were based on studies completed in the last decade with 34 different organic and inorganic chemicals. Embryo-larval and early juvenile life stages were found to be the most, or among the most, sensitive. Tests with these life stages apparently could be used to estimate the maximum acceptable toxicant concentration (MATC) within a factor of 2 in most instances. Hamelink and Spacie[20] presented an overview of papers dealing with how fish acquire chemical residues, pharmacokinetic models used to predict bioaccumulation in fish, and fish growth and biomass dynamics.

After completing their review the authors concluded that the environmental behavior of pollutants appeared to depend more on their chemical-physical properties than on the biological-ecological features of the receiving waters.

The effects of Al on various aquatic organisms, its chemistry in dilute aqueous solutions, and its occurrence in natural waters were reviewed by Burrows.[21] Of the 225 papers reviewed, 20 were concerned with the acute and chronic toxicity of Al to freshwater fishes. The EIFAC [22] prepared a Cu report, which outlined laboratory and field findings regarding its toxicity to fish, principally salmonids, and other aquatic organisms. It was reported that the toxicity of Cu was increased when water hardness, temperature, dissolved oxygen, chelating agents, humic acids, amino acids, and suspended solids were reduced. An extensive review of Cr including information on its chemical and physical properties, its distribution in the environment and toxicological data in fish and other animals including metabolism, toxicity, mutagenicity, teratogenicity, and carcinogenicity was presented by Towell *et al.*[23] The U. S. Environmental Protection Agency (EPA)[24] reviewed and evaluated existing monitoring data on Cd. Their report reviewed environmental levels of Cd based on published reports and other information sources. The highest level of Cd found in fish (taken from the Columbia River) was 1.7 mg/l. Marine fish reportedly contained less Cd than freshwater fish. In the water-based food chain, pond fish apparently contained less Cd than stream fish. In reviewing the literature on Cd, the EIFAC [25] stated that acute and chronic toxicity tests with sensitive species of fish have yielded conflicting results. Speculation as to the causes of such variation were given. Hammons *et al.*[26] prepared an extensive review of the environmental effects of Cd for EPA. In addition to consideration of the effects of Cd on fish and other organisms, the review included information on chemical and physical properties, analytical techniques, transport processes, and human health effects. The review concluded with an assessment of the effects of Cd in the environment and an analysis of research needs. Taylor [27] summarized the acute and sublethal effects of Cd on both marine and freshwater organisms, including fish, based on 122 references. Another document prepared by Taylor [28] summarized over 100 scientific papers dealing with the acute and sublethal effects of Hg on marine and freshwater organisms. A review of Hg in the environment based on published data and other sources of information was also prepared by EPA.[29] Several tables were presented summarizing the Hg content of fishes collected from different geographical areas of the U. S. Levels of Hg in the tissues of aquatic biota, both freshwater and marine, appeared to be consistently higher in the industrialized Atlantic coast region. The report discussed bioaccumulation of Hg in the aquatic food chain and the occurrence of Hg in commercially important species of fish. Some environmental and experimental effects (fish mentioned) of Hg and some areas for future research were outlined by the World Health Organization.[30] Taylor [31] prepared a bibliography of some 4 000 references covering all aspects of environmental pollution by Hg. The references were selected from the literature published prior to June of 1977. Subject matter ranged from analysis of Hg compounds to biological effects. Eisler *et al.*[32] listed 954 titles of technical articles on the subject of toxicological, physiological, and metabolic effects of stable and radio-labeled chemical species of metal cations to marine, estuarine, and freshwater flora and fauna. Each reference was annotated and subsequently indexed by metal, taxa, and author.

Newton and Norgren [33] prepared a comprehensive review on management practices involving silvicultural chemicals. The report provided a technical review of the toxicological properties of important pesticides and chemicals used in forest management and described the scope of usage and the effects of these management tools on water quality, aquatic animals, and related resource values. The literature pertaining to the acute and chronic effects of pesticides on aquatic organisms was reviewed by Livingston.[34] The literature was organized into three general categories: physiochemical characteristics, use and relative toxicity, and the overall impact of pesticides on aquatic animals. The nature and origins of pollution of aquatic systems by pesticides were reviewed by Edwards.[35] Persistence, toxicity, and bioconcentration, as well as levels of pesticides (especially the organochlorine insecticides and certain herbicides) in fish and other organisms, were considered. A book entitled "Pesticides in Aquatic Environments" was edited by Khan [36] dealing specifically with the physiochemical and biochemical dynamics of pesticides in aquatic environments. The contributors thoroughly covered metabolic path-

ways of pesticides and other environmental contaminants in aquatic organisms, including fish, as well as their toxicity to these animals. Other topics discussed included: release and transport of pesticides, residues, uptake, and biodegradability of pesticides in model ecosystems. The proceedings of a workshop to update data on pesticides in Iowa lakes and streams and to identify areas needing research was published by the Iowa State Water Resources Institute.[37] Craig [38] summarized data from over 230 papers covering lethal and sublethal effects of aldrin, dieldrin, and endrin on aquatic life. Reported residue levels in aquatic animals were also presented. Mehrle *et al.*[39] summarized the results of four published toxicological studies, conducted at the Fish-Pesticide Research Laboratory, which illustrated that nutritional factors influenced the effects of chlordane, DDT, dieldrin, toxaphene, and PCBs on fish. Russel-Smith [40] gave preliminary observations of the effects of endosulfan application on the aquatic fauna of the Okavango Delta in Botswana. The author reviewed the general effects of insecticides on aquatic animals and discussed, in particular, endosulfan and its effects on fish and aquatic invertebrates. Data were also given on endosulfan residues in the Shoreobe lagoon. The history of the use and findings of the toxicity, persistence, and the control of mirex and related compounds were reviewed by Kaiser.[41] Field studies showed that mirex contaminated the biota of Lake Ontario, and the results of sediment studies indicated that the Niagara River was the major contributor of mirex. Much of the widespread contamination of aquatic birds in the Great Lakes area was found to come from mirex in Lake Ontario water and fish. In a report prepared for EPA,[42] the National Research Council assessed the scientific and technical knowledge concerning the effects of kepone, mirex, and hexachlorocyclopentadiene as environmental pollutants. The principal findings were summarized in regard to distribution, transport and fate, exposure to humans, and environmental considerations. The use of biological indicator organisms to monitor the comparative pollution of aquatic areas by organochlorine compounds was reviewed by Phillips.[43] It was determined that numerous parameters, such as age, size, and sex of the organisms sampled, can influence survey results. Zitko [44] reviewed data from previous papers on the uptake and excretion of PCBs and PBBs, bromobiphenyls and chlorobiphenyls, isopropylated chlorobiphenyls (Chloralkylene 12), chlorinated diphenyl ethers, brom-, and bromochloro-substituted benzenes and toluenes administered to juvenile Atlantic salmon (*Salmo salar*) in water and in food. Accumulation coefficients and excretion half-lives were calculated for these halogenated aromatic hydrocarbons from both water and food exposures. Comparative aspects of uptake and excretion of halogenated aromatic hydrocarbons in exposed fish were discussed.

In preparing an inventory of environmental contaminants, Leah [45] summarized some of the pertinent literature on the subjects of environmental levels of phthalates and the toxicity of phthalates to biota including fish. Accumulation, metabolism, excretion, and effect of phthalates on reproduction were also reviewed. Information on production, use, transport, handling of environmental fate, and the toxicity of benzotriazoles to several fish including trout, sunfish, and minnows was reviewed by Davis *et al.*[46] Several references pertaining to the toxicity and bioaccumulation of benzidine in fish were listed by Shiner *et al.*[47] Burg *et al.*[48] reported that seven different fluorescent whitening agents are currently used in American-manufactured detergents. According to the literature they surveyed, fluorescent whitening agents were identified routinely in wastewater. Only trace amounts appeared in drinking water and all compounds had low fish toxicity but a high bioconcentration potential. A literature review pertaining to the manufacture and waste disposal of paper and allied products was compiled by Gove and Gellman.[49] Many subjects ranging from policies and economics to effluent toxicity and characterization were covered. In addition, papers on the physiochemical treatment of effluent were reviewed.

Mazeaud *et al.*[50] discussed the effects of stress, such as struggling, hypoxia, heat, capture, and transportation on the endocrine systems of fish, especially salmonids. These types of stress resulted in significant increases in plasma catecholamine and corticosteroid concentrations. The authors also reviewed some metabolic changes that occur as a result of endocrine changes and found that metabolic disturbances include a pronounced increase in blood glucose, and either a decrease or an increase in plasmatic-free fatty acids dependent on the fish species tested. According to Harshbarger,[51] the Registry of Tumors in Lower Animals (Smithsonian Institution) has served for a decade as a diagnostic center, literature

center, specimen depository, and a clearinghouse for information on neoplasia and related disorders in invertebrates and cold-blooded animals. Based on the registry's literature file, Harshbarger prepared a list of animal populations, including fish, from which neoplasms have been repeatedly found since 1966. Carcinogens that reportedly can experimentally induce neoplasm and hyperplasia in fish and other lower animals were also listed. In their review of neoplasms in rainbow trout (*Salmo gairdneri*), Sinnhuber *et al.*[52] pointed out that rainbows are the more sensitive to aflatoxin carcinogenicity than any other known group of animals or species of fish. Most of their discussion centered around AFB_1 carcinogenesis. It was noted, however, that rainbows responded in a similar manner (same types of lesions) to different types of hepatic carcinogens. It was concluded that more information was needed, particularly on the pathogenesis of rainbow trout liver tumors. Morgan [53] prepared a short review of the physiological and behavioral responses of fish that may prove useful as endpoints for biomonitoring the quality of surface waters and effluents.

WATER QUALITY

Dissolved Gases. Overholtz *et al.*[54] monitored the effects of oxygenation on depth distribution of rainbow trout (*Salmo gairdneri*) and gizzard shad (*Dorosoma cepedianum*) after hypolimnion water was withdrawn from a quarry pond, oxygenated, and returned without upsetting thermal stratification. Summer DO concentrations were raised from almost 0 to 8 mg/l in 1973 and ranged from 7.0 to 21.5 mg/l in 1974. Stocked rainbow trout, previously unable to survive summer temperatures and low DO levels, were found distributed near the upper boundary of the hypolimnion and apparently suffered no ill effects from DO concentrations as high as 21.5 mg/l. Boyd *et al.*[55] studied DO problems in catfish ponds and determined that:

- Fish kills occurred in only two of five instances following phytoplankton die-offs which resulted in dangerously low DO levels (< 1 mg/l).
- A computer simulation model predicted nighttime DO declines within ±10% of measured concentrations in ponds.
- Neither potassium permanganate nor simazine should be applied to ponds in an attempt to alleviate critically low DO levels or reduce phytoplankton density.

Brooker *et al.*[56] concluded that reported major fish kills in the River Wye (Wales) were precipitated by rapidly increasing temperatures associated with rapidly decreasing DO concentrations. The DO levels, which fell as low as 0.5 mg/l, were caused by sunny and low flow conditions that accelerated the decay of substantial plant stands and were considered the dominant factor in producing the mass mortalities. Declines in benthic fish populations in Oneida Lake (New York) were attributed by Clady [57] to abiotic factors, to particularly worsening DO conditions in deeper waters, and to wind velocity because of its moderating effects on thermal stratifications and DO depletion. Abundance of year-classes of trout-perch (*Percopsis omiscomaycus*) and tessellated darters (*Etheostoma olmstedi*) was poor in 1970, 1972, and 1973 when DO levels dropped as low as 3.3, 0.4, and 1.7 mg/l, respectively, and in calmer summers when mean wind velocities dropped to 3.3 to 3.8 m/s. Tramer [58] studied catastrophic mortality of fishes trapped in small stream-bed pools, produced during summer drought, and found that pulses of heavy diurnal mortality were generally correlated with low DO levels in the early morning. From studies in the small Baise River, Lim [59] reported increased survival time of *Cyprinidae* exposed in immersion experiments to river water containing ammonia and hydrazine. These results verified that the improvement works undertaken by the Kuhlmann factory at Lannemezan resulted in a 50% decrease in the ammonical N, a 66% decrease in the hydrazine, and a general decrease in toxic wastes of about 50% over a 2-year period.

By evaluating the effects of gas supersaturated water on steelhead trout (*Salmo gairdneri*), Nebeker *et al.*[60] showed that eggs, embryos, and newly hatched larvae were not affected at 126.7% saturation. However, onset of gas bubble disease occurred at swim-up (16 days posthatch), and mortality ranged from 99% at 126.7% saturation to 45% at 115.5% saturation. No differences in survival were noted between fish exposed from egg to fry, and those exposed only from swim-up to fry stage. Mountain whitefish (*Prosopium williamsoni*) were the most sensitive of four species tested by Fickeisen and Montgomery [61] for tolerance to gas supersaturation in deep tank bioassays. Median times to death (LT-50) for this species ranged from 12 hours at 128% saturation to 48 hours at 116%. Following in order of decreasing sensitivity, were cutthroat trout (*Salmo clarki*), large-scale

sucker (*Catostomus macrocheilus*), and torrent sculpin (*Cottus rhotheus*). The latter required 10 days to reach an 50% lethal time (LT50) at 128% saturation, had no deaths at 116%, and typically developed large gas bubbles that caused positive buoyancy and indirectly contributed to death. A five-phase study by Weitkamp [62] related laboratory studies and existing standards concerned with air supersaturation to the river environment. In addition to a thorough literature review, resident fish populations in the Columbia River system were sampled and analyzed; the depth distribution of migrating juvenile salmon was studied; and bioassays using juvenile salmonids were conducted in the river environment. These studies showed that when provided water 4 m deep, few fish developed gas bubble disease, no mortalities occurred within 20 days at 120 to 130% TGP, and that compensation by suitable hydrostatic pressure was a survival mechanism. Matthews and Hill [63] documented the responses of the red shiner (*Notropis lutrensis*) to changes in DO, temperature, salinity, and pH. This species showed stress but survived a DO concentration of 1.5 mg/l, and died at 1.0 mg DO/l without an atmosphere. The fish also survived hydrogen ion concentrations of 5 to 10 pH units, salinity concentrations of 10 ppt, and thermal shocks of T + 10° to T − 21°C. Carlson and Herman [64] determined the spawning success of the black crappie (*Pomoxis nigromaculatus*) exposed to diel fluctuating DO concentrations after overwinter exposure to reduced DO regimes. It was concluded that DO concentrations near 2.6 mg/l or above satisfied black crappie DO requirements during these periods. Diel fluctuations below 2.6 to 1.8 mg/l, during simulated spring water temperature rises, caused hypoxic stress and inhibited reproduction.

Wedemeyer and Yasutake [65] investigated the efficacy of mineral salts and pH adjustments in reducing the toxicity of nitrite to hatchery-reared steelhead trout, and the effectiveness of methylene blue in treating fish suffering from methemoglobinemia. The acute toxicity of nitrite to fingerling steelhead trout in static, renewal tests was reduced by a factor of 24 to 13, depending on the size of the fish, when total water hardness was increased from 25 to 300 μg/l. Increase in pH and addition of NaCl and $CaCl_2$ also reduced nitrite toxicity. Thurston *et al.*[66] found that cutthroat trout fry susceptibility to acute toxicity from aqueous ammonia and nitrite was comparable to previously determined values for rainbow trout. The 96-hour 50% lethal concentration (LC50) values for the cutthroat were 0.5- to 0.8-mg/l unionized ammonia and 0.5- to 0.6-mg nitrite/l, and for 36 days were 0.3- to 0.6-mg ammonia/l and 0.4-mg nitrite/l. Additionally, histological examination of fish exposed for 29 days to 0.34-mg ammonia/l showed degenerative changes in gill, kidney, and liver tissue. Rubin and Elmaraghy [67] determined the individual and joint toxicity of ammonia and nitrate of guppy fry (*Poecilia reticulata*), and evaluated the applicability of Chick's law and other kinetic models to fish toxicity. The 72-hour LC50 values were 199 and 1.26 mg/l for potassium nitrate and free ammonia, respectively. Except at very low ammonia to nitrate ratios, mixture toxicity was additive. A disinfection law relating toxicant concentration to survival, Watson's equation, was found applicable to both toxicants. In other ammonia bioassays, the 96-hour LC50 for coho salmon (*Oncorhynchus kisutch*), exposed by Buckley [68] to unionized ammonia, was 0.45 mg/l and the highest concentration tested with no acute effects was 0.178 mg/l. While conducting tests on water reuse systems, Fyock [69] determined that the tolerance limits of ammonia (0.55 mg/l) and nitrite (0.2 mg/l) for rainbow trout were not exceeded by the ammonia produced from 0.94 kg of 20-cm trout/m^2 or 1.34 kg of 35-cm trout/m^2 of surface·d in a racewater providing 216 m^2 of nitrifying surface area in a biological filtration system. Nor was nitrification affected by the therapeutic compounds Hyamine-3500 at 1.0 mg/l or Malachite green at 3.0 mg/l. However, the Malachite green caused 100% fish mortality.

Broderius *et al.*[70] defined the change in tolerance limits of the fathead minnow (*Pimephales promelas*) to free cyanide and dissolved sulfide forms (see Table I) as a function of pH, and related the bioassay results to proposed gill permeability theories. Smith *et al.*[71] reported on 337 acute toxicity tests designed to determine the 96-hour LC50 and the median lethal threshold concentration (LTC) of hydrogen cyanide (HCN) at temperatures between 4° to 30°C and DO concentrations from 3.36 to 9.26 mg/l for three life history stages of four fish species and for juveniles of one other (Table I). Acute toxicity among juvenile fish, the most sensitive stage along with newly hatched fry, varied from 57 μg/l for rainbow trout to 191 μg/l for field stocks of fathead minnows. Juvenile fish were also more sensitive at lower temperatures

TABLE I. Summary of the acute and chronic toxicity of inorganic and organic pollutants to freshwater fish.

Pollutant	Species	Results	Exposure Type[a]	Temp. (°C)	Test Conditions
Abate (50% EC)[127]	Chingatta (*Channa gachua*)	96-h LC50 217 mg/l	S[a]	24	DO[b] 5.0–7.2 HD[c] 60 pH 7.2
Abietic resin acid[400]	Coho salmon (*Oncorhynchus kisutch*) Juvenile	96-h LC50 0.41 mg/l	R[d]	—	—
Abietol[400]	Rainbow trout (*Salmo gairdneri*) Juvenile	96-h LC50 1.8 mg/l	S	—	—
Acetone[300]	Rainbow trout	24-h LC50 6 100 mg/l	F[e]	10	HD 90 pH 8.0
Aerozine-50[313]	Guppy (*Poecilia reticulata*)	24-h LC50 12.3 mg/l	S	22–24.5	ALK[f] 185–232
		48-h LC50 4.37 mg/l	S	22–24.5	DO 6.9–7.8
		72-h LC50 2.72 mg/l	S	22–24.5	HD 400–500
		96-h LC50 2.25 mg/l	S	22–24.5	pH 7.8–8.2
		24-h LC50 5.08 mg/l	S	22–24.5	ALK 16–18
		48-h LC50 2.86 mg/l	S	22–24.5	DO 6.9–7.8
		72-h LC50 1.95 mg/l	S	22–24.5	HD 20–25
		96-h LC50 1.17 mg/l	S	22–24.5	pH 7.8–8.2
Alfatoxin B_1[316]	Japanese medaka (*Oryzias latipes*) Eggs	720-h LC50 0.18 μg/ml	S	22.1	pH 7.1
Aflatoxin B_1[346]	Japanese medaka Eggs	720-h LC50 0.18 μg/ml	—	—	—
alamine 336[210]	Rainbow trout	96-h LC50 7.5–10 mg/l	S	15	HD 43 pH 8.5
Aliquat 336[210]	Rainbow trout	96-h LC50 0.18–0.32 mg/l	S	15	HD 43 pH 8.5
Altosid[124]	Coho salmon	96-h LC50 86 mg/l	S	11	ALK 2.2
	Rainbow trout	96-h LC50 106 mg/l	S	11	HD 5 pH 6.5
Amberlite LA-1[210]	Rainbow trout	96-h LC50 0.25 mg/l	S	15	HD 43 pH 8.5
4-Amino-3,5 xylenol (99%)[120]	Bluegill (*Lepomis macrochirus*)	96-h LC50 0.32 mg/l	S	12	ALK 30–35 HD 40–48 pH 7.5
		96-h LC50 0.087 mg/l	S	12	HD 40–48 pH 9.5
Ammonia[67]	Guppy Fry	72-h LC50 74 mg/l	S	25–26.7	ALK 25.7–33.2 DO 6.8–8.2 HD 128–194 pH 7.4–7.5
Ammonia (free ammonia)[67]	Guppy Fry	72-h LC50 1.26 mg/l	S	25–26.7	ALK 25.7–33.2 DO 6.8–8.2 HD 128–194 pH 7.4–7.5
Ammonia (unionized)[66]	Cutthroat trout (*Salmo clarki*) Fry	96-h LC50 0.5–0.8 mg/l	F	11.7–14.7	ALK 178 DO 7.6–9.2
		36-d LC50 0.56 mg/l	F	11.7–14.7	HD 200 pH 7.8

TABLE I—(*Continued*)

Pollutant	Species	Results	Exposure Type	Temp. (°C)	Test Conditions
Ammonia (unionized)[48]	Coho salmon	96-h LC50 0.45 mg/l	F	14.7–19.7	ALK 38–136 DO >80% Saturation HD 37 pH 7.12–9.10
Bayer 73 (70% WP)[183]	Sea lamprey (*Petromyzon marinus*) Larvae	96-h LC50 0.030–0.097 mg/l	S	7–17	HD 44–300 pH 6.5–8.5
	Burrowed larvae	96-h LC50 0.129 mg/l	F	12	—
	Free swimming larvae	96-h LC50 0.034 mg/l	F	12	—
	Rainbow trout	96-h LC50 0.018 mg/l	F	12	—
	Brook trout (*Salvelinus fontinalis*)	96-h LC50 0.034 mg/l	F	12	—
3,4-Benzopyrene[331]	Atlantic salmon (*Salmo salar*) Eggs	168-h BCF[a] 70.7	S	4	—
Bromine chloride[200]	Fathead minnow (*Pimephales promelas*)	96-h LC50 0.133–0.193 mg/l	F	25	—
	Pugnose shiner (*Notropis anogenus*)	96-h LC50 0.109–0.136 mg/l	F	25	—
	Common shiner (*Notropis cornutus*)	96-h LC50 0.120–0.140 mg/l	F	25	—
	Golden shiner (*Notemigonus crysoleucas*)	96-h LC50 0.090 mg/l	F	25	—
Bromine chloride[200]	Yellow bullhead (*Ictalurus natalis*)	96-h LC50 0.177 mg/l	F	25	—
	Black bullhead (*Ictalurus melas*)	96-h LC50 0.283 mg/l	F	25	—
	Lake trout (*Salvelinus namaycush*)	96-h LC50 0.102 mg/l	F	14	—
	Chinook salmon (*Oncorhynchus tschawytscha*)	96-h LC50 0.059 mg/l	F	14	—
Cadmium (cadmium nitrate)[215]	Mosquitofish (*Gambusia affinis*)	96-h LC50 1.3 mg/l 96-h LC50 1.5 mg/l 96-h LC50 2.6 mg/l	F F F	30.2 30.7 28.0	ALK 4.0 DO >90% Saturation HD 10.0 pH 5.6
		96-h LC50 0.9 mg/l 96-h LC50 2.2 mg/l	F F	30.2 28.0	ALK 9.7 DO >90% Saturation HD 11.1 pH 6.5
Cadmium (cadmium chloride)[220]	Goldfish (*Carassius auratus*)	48-h LC50 2.76 mg/l 96-h LC50 2.13 mg/l	S S	22.0–25.4 22.0–25.4	ALK 14–18 DO >90% Saturation
		240-h LC50 1.78 mg/l	S	22.0–25.4	HD 20–22
		48-h LC50 46.9 mg/l 96-h LC50 46.8 mg/l 240-h LC50 40.2 mg/l	S S S	22.0–25.4 22.0–25.4 22.0–25.4	DO 8 HD 143–145

TABLE I—(*Continued*)

Pollutant	Species	Results	Exposure Type	Temp. (°C)	Test Conditions
Cadmium (cadmium chloride)[327]	White sucker (*Catastomus commersoni*) Larvae-juvenile	30-d MATC[a] 4.2–12.0 µg/l (preceded by 10-d embryo exposure)	F	18.1	A[i] 3.0 ALK 41 DO 10.3 HD 45 pH 7.6
	Northern pike (*Esox lucius*) Larvae-juvenile	28-d MATC 4.2–12.9 µg/l (preceded by 7-d embryo exposure)	F	15.9	
	Smallmouth bass (*Micropterus dolomieui*) Larvae-juvenile	30-d MATC 4.3–12.7 µg/l (preceded by 3-d embryo exposure)	F	15.9	
	Coho salmon Larvae-juvenile	27-d MATC 1.3–3.4 µg/l (begun with sac fry)	F	10.1	
		62-d MATC 4.1–12.5 µg/l (preceded by 20-d embryo exposure)	F	9.7	
	Lake trout Larvae-juvenile	64-d MATC 4.4–12.3 µg/l (preceded by 10-d embryo exposure)	F	9.6	
	Brook trout Larvae-juvenile	65-d MATC 1.1–3.8 µg/l (preceded by 24-d embryo exposure)	F	9.7	
	Brown trout (*Salmo trutta*) Larvae-juvenile	60-d MATC 3.8–11.7 µg/l (preceded by 50-d embryo exposure)	F	9.7	
	Brown trout Larvae-juvenile	61-d MATC 1.1–3.7 µg/l (preceded by 2-d exposure, late eyed embryos)	F	10.0	
Carbaryl (50% WP)[138]	*Channa punctatus*	180-d LC50 2.0 mg/l	R	—	—
Chlordane[126]	*Saccobranchus fossilis*	96-h LC50 0.42 mg/l	S	—	—
Chlorine[379]	Goldfish	24-h LC50 0.49 mg/l	F	20–22.5	HD 45–96
		48-h LC50 0.38 mg/l	F	20–22.5	pH 7.09–9.25
		72-h LC50 0.35 mg/l	F	20–22.5	
		96-h LC50 0.35 mg/l	F	20–22.5	
Chlorine[383]	Coho salmon Alevin	96-h LC50 80–83 µg/l	F	9.9–10.2	ALK 135–180
	Fry	96-h LC50 79 µg/l	F	10.3	pH 7.0–8.2
	Juvenile	96-h LC50 53–82 µg/l	F	10.5–15.0	
	Brook trout Alevin	96-h LC50 90.6–105.5 µg/l	F	10.6–10.8	
	Fry	96-h LC50 81.8 µg/l	F	10.8	
	Juvenile	96-h LC50 88.4–90.6 µg/l	F	11.1–11.3	
	Cutthroat trout Juvenile	96-h LC50 74.5–94.7 µg/l	F	10.1–15.1	
Chlorine[380]	Coho salmon	30-min LC50 0.56 mg/l	S	10.3	DO 7.4–10.9
		30-min LC50 0.29 mg/l	S	20.1	pH 8.23–8.53
	Alewife (*Alosa pseudoharengus*)	30-min LC50 2.15 mg/l	S	10.5	
		30-min LC50 2.27 mg/l	S	15.2	
		30-min LC50 1.70 mg/l	S	20.1	
		30-min LC50 0.96 mg/l	S	25.1	
		30-min LC50 0.30 mg/l	S	29.8	
	Spottail shiner (*Notropis hudsonius*)	30-min LC50 2.41 mg/l	S	10.1	
		30-min LC50 1.00 mg/l	S	15.1	
		30-min LC50 0.53 mg/l	S	20.2	

TABLE I—(*Continued*)

Pollutant	Species	Results	Exposure Type	Temp. (°C)	Test Conditions
	Rainbow smelt (*Osmerus mordax*)	30-min LC50 1.27 mg/l	S	9.9	
Chlorine[202]	Brook trout Alevin	96-h LC50 105.5 μg/l	F	10.6	DO 8.6–10.0
	Alevin	96-h LC50 90.6 μg/l	F	10.8	HD 130–150
	Fry	96-h LC50 82.0 μg/l	F	10.6	pH 7.7–7.8
	Juvenile	96-h LC50 90.6 μg/l	F	11.3	
	Juvenile	96-h LC50 88.4 μg/l	F	11.1	
Chlorine[203]	Fathead minnow	96-h LC50 0.082–0.095 mg/l	F	25	—
	Pugnose shiner	96-h LC50 0.045 mg/l	F	25	—
	Common shiner	96-h LC50 0.051 mg/l	F	25	—
	Golden shiner	96-h LC50 0.040 mg/l	F	25	—
	Goldfish	96-h LC50 0.153–0.210 mg/l	F	25	—
	Lepomis sp.	96-h LC50 0.195–0.278 mg/l	F	25	—
	Pomoxis sp.	96-h LC50 0.127 mg/l	F	25	—
	Largemouth bass (*Micropterus salmoides*)	96-h LC50 0.241 mg/l	F	25	—
	Walleye (*Stizostedion vitreum vitreum*)	96-h LC50 0.108 mg/l	F	25	—
	Lake trout	96-h LC50 0.060 mg/l	F	14	—
	Rainbow trout	96-h LC50 0.069 mg/l	F	—	—
	Coho salmon	96-h LC50 0.059 mg/l	F	—	—
Chlorine (chloramine)[270]	Golden shiner	96-h LC50 0.724 mg/l	F	6	DO Near Saturation
		96-h LC50 0.930 mg/l	F	24	HD 45
	Carp (*Cyprinus carpio*)	96-h LC50 1.72 mg/l	F	6	pH 6.8–7.7
	Coho salmon	96-h LC50 0.677 mg/l	F	6	
		96-h LC50 0.640 mg/l	F	12	
Chlorine (free)[270]	Golden shiner	96-h LC50 0.269 mg/l	F	6	DO Near Saturation
		96-h LC50 0.193 mg/l	F	24	HD 45
	Carp	96-h LC50 0.538 mg/l	F	6	pH 7.0–7.6
		96-h LC50 0.219 mg/l	F	24	
	Channel catfish (*Ictalurus punctatus*)	96-h LC50 0.082 mg/l	F	6	
	Coho salmon	96-h LC50 0.064 mg/l	F	24	
		72-h LC50 0.181 mg/l	F	6	
		72-h LC50 0.119 mg/l	F	12	
4-Chloroaniline[135]	Rainbow trout	96-h LC50 14 mg/l	S	12	ALK 35
	Fathead minnow	96-h LC50 12 mg/l	S	22	HD 40
	Channel catfish	96-h LC50 23 mg/l	S	22	pH 7.2
	Bluegill	96-h LC50 2 mg/l	S	22	
5-Chloro-2-methyl-4-isothiazolin-3-one calcium chloride[140]	Bluegill	49-d BCF 22–27 (whole fish minus viscera)	F	18	DO >5.0 HD 25
		49-d BCF 157–300 (viscera)	F	18	pH 6.8
		43-d BCF 30 (whole fish minus viscera)	F	18	
		43-d BCF 204 (viscera)	F	18	
4-Chlorophenyl urea[135]	Rainbow trout	96-h LC50 72 mg/l	S	12	ALK 35
	Fathead minnow	96-h LC50 >100 mg/l	S	22	HD 40
	Channel catfish	96-h LC50 >100 mg/l	S	22	pH 7.2
	Bluegill	96-h LC50 >100 mg/l	S	22	

TABLE I—(*Continued*)

Pollutant	Species	Results	Exposure Type	Temp. (°C)	Test Conditions
Chromium (potassium chromate)[317]	Goldfish	24-h LC50 109–354 mg/l	S	5–30	DO 6–11
	Golden shiner	24-h LC50 104–151 mg/l	S	5–30	HD 36
	Bluegill	24-h LC50 214–280 mg/l	S	5–30	pH 5.8
	Channel catfish	24-h LC50 50–72 mg/l	S	5–30	
	Rainbow trout	24-h LC50 59–141 mg/l	S	5–18	
Coal-conversion gasifier condensate[431]	Fathead minnow	96-h LC50 0.051%	R	18	pH 7.8–8.7
		7-d LC50 0.012%	R	18	
Copper[279]	Goldfish	24-h LC50 1.14 mg/l	F	20–22.5	HD 44.9–96.0
		48-h LC50 0.60 mg/l	F	20–22.5	pH 7.09–9.25
		72-h LC50 0.30 mg/l	F	20–22.5	
		96-h LC50 0.30 mg/l	F	20–22.5	
Copper[217]	Goldfish	24-h LC50 1.5–2.9 mg/l	S	5–30	DO 6–11
	Golden shiner	24-h LC50 0.2–0.3 mg/l	S	5–30	HD 36
	Bluegill	24-h LC50 2.5–3.8 mg/l	S	5–30	pH 7.2
	Channel catfish	24-h LC50 2.6–3.7 mg/l	S	5–30	
	Rainbow trout	24-h LC50 0.2–1.0 mg/l	S	5–18	
Copper[216]	Rainbow trout	96-h LC50 19.9–30.0 µg/l	F	15	DO >90% Saturation HD 30–32 pH 5.0–9.0
		96-h LC50 33.1–176 µg/l	F	15	DO >90% Saturation HD 98–102 pH 4.9–9.0
		96-h LC50 70.0–516 µg/l	F	15	DO >90% Saturation HD 360–371 pH 5.0–9.0
Copper (copper sulfate)[329]	Rainbow trout Larvae-juvenile	35-d MATC 11.4–31.7 µg/l (preceded by 11-d embryo exposure)	F	10.8	A 1.9 ALK 42.4 DO 11.4 HD 45.4 pH 7.3–7.9
	White sucker Larvae-juvenile	27-d MATC 12.9–33.8 µg/l (preceded by 13-d embryo exposure)	F	14.9	
	Northern pike Larvae-juvenile	34-d MATC 34.9–104.4 µg/l (preceded by 6-d embryo exposure)	F	15.6	
	Brook trout Larvae-juvenile	60-d MATC 22.3–43.5 µg/l (preceded by 16-d embryo exposure)	F	5.6	
	Brown trout Larvae-juvenile	55-d MATC 22.0–43.2 µg/l (preceded by 72-d embryo exposure)	F	5.7	
	Lake trout Larvae-juvenile	66-d MATC 22.0–42.3 µg/l (preceded by 27-d embryo exposure)	F	5.5	
Copper (copper sulfate)[230]	Fathead minnow Larvae-juvenile	96-h LC50 490 µg/l	F	20.0–24.5	A 8 DO 7.8
		96-h LC50 460 µg/l	F	20.0–24.5	HD 202
		6-mo MATC 32 µg/l	F	20.0–24.5	pH 7.5–8.2
Cyanide[217]	Goldfish	24-h LC50 0.3–3.3 mg/l	S	5–30	DO 6–11
	Golden shiner	24-h LC50 0.3–0.5 mg/l	S	5–30	HD 36
	Bluegill	24-h LC50 0.2 mg/l	S	5–30	pH 7.6
	Channel catfish	24-h LC50 0.2–0.3 mg/l	S	5–30	
	Rainbow trout	24-h LC50 0.1 mg/l	S	5–18	

TABLE I—(*Continued*)

Pollutant	Species	Results	Exposure Type	Temp. (°C)	Test Conditions
Cyanide (free cyanide)[70]	Fathead minnow Juvenile	96-h LC50 117–157 µg/l	F	20	ALK 96–250 DO 7.56–7.71 Free CO_2 0.24–28.0 HD 220 pH 6.8–9.3
Cyanide (hydrogen cyanide)[70]	Fathead minnow Juvenile	96-h LC50 83–124 µg/l	F	20	ALK 96–250 DO 7.56–7.71 Free CO_2 0.24–28.0 HD 220 pH 6.8–9.3
Cyanide (hydrogen cyanide)[71]	Fathead minnow, Eggs	96-h LC50 121–352 µg/l	F	15–25	DO 3.5–7.3
	Swim up fry	96-h LC50 82–122 µg/l	F	15–25	HD 220
	Juvenile	96-h LC50 82–137 µg/l	F	15–25	pH 7.7–8.2
	Juvenile (wild stock)	96-h LC50 157–191 µg/l	F	15–30	
Cyanide (hydrogen cyanide)[71]	Bluegill, Eggs	96-h LTC 535–693 µg/l (Lethal threshold concentration)	F	25	DO 3.4–8.4 HD 220 pH 7.7–8.1
	Swim up fry	96-h LC50 232–365 µg/l	F	20–25	
	Juvenile	96-h LC50 75–125 µg/l	F	8.4–25.1	
	Yellow perch (*Perca flavescens*) Eggs	96-h LC50 >276–>389 µg/l	F	14–18	DO 3.4–7.4
	Swim up fry	96-h LC50 295–>395 µg/l	F	10–18	HD 220
	Juvenile	96-h LC50 76–108 µg/l	F	15–21	pH 7.7–7.9
	Brook trout, Eggs	96-h LC50 >212–>242 µg/l	F	7–13	DO 3.5–9.3
	Sac fry	96-h LC50 108–518 µg/l	F	10–13	HD 220
	Swim up fry	96-h LC50 56–106 µg/l	F	7–13	pH 7.7–8.1
	Juvenile	96-h LC50 53–143 µg/l	F	4–18	
	Rainbow trout	96-h LC50 57 µg/l	F	10	DO 8.8
Dalapon[177]	*Rasbora trilineata*	96-h LC50 135 mg/l	S	—	—
	Guppy	96-h LC50 223 mg/l	S	—	—
Decamethrin[132]	Rainbow trout	48-h LC50 0.5 µg/l	S	—	—
	Desert Pupfish (*Cyprinodon macularius*)	48-h LC50 0.6 µg/l	S	—	—
	Mosquitofish	48-h LC50 1.0 µg/l	S	—	—
	Tilapia mossambica	48-h LC50 0.8 µg/l	S	—	—
Dehydroabietic resin acid[406]	Coho salmon Juvenile	96-h LC50 0.75 mg/l	R	—	—
Dehydroabietol[406]	Rainbow trout	96-h LC50 0.8 mg/l	S	—	—
Diazinon[146]	Brook trout	96-h LC50 0.45–1.05 mg/l	F	12	—
		240-d MATC 3.2 µg/l	F	9–15	A 3.4 ALK 42 HD 45 pH 7.3
	Fathead minnow	96-h LC50 6.8–10.0 mg/l	F	25	—
		274-d MATC 3.2 µg/l	F	25	A 2.9 ALK 42 HD 44 pH 7.5

TABLE I—(*Continued*)

Pollutant	Species	Results	Exposure Type	Temp. (°C)	Test Conditions
	Flagfish (*Jordanella floridae*)	96-h LC50 1.5–1.8 mg/l	F	25	—
	Bluegill	96-h LC50 0.44–0.48 mg/l	F	25	—
Dichlorodehydro-abietic acid[408]	Rainbow trout Juvenile	96-h LC50 0.6 mg/l	S	—	—
2,4-D (butylthio)[172]	Mosquitofish	48-h LC50 0.98 mg/l (susceptible population)	S	24	—
		48-h LC50 1.70 mg/l (resistant population)	S	24	—
2,4-D (ethylthio)[172]	Mosquitofish	48-h LC50 1.34 mg/l (susceptible population)	S	24	—
		48-h LC50 1.58 mg/l (resistant population)	S	24	—
2,4-Dichlorophenyl p-nitrophenyl ether[177]	*Rasbora trilineata*	96-h LC50 0.756 mg/l	S	—	—
	Guppy	96-h LC50 1.59 mg/l	S	—	—
9,10 Dichloro-stearic acid[408]	Rainbow trout Juvenile	96-h LC50 2.5 mg/l	S	—	—
2,6 Difluorobenzoic acid[135]	Rainbow trout	96-h LC50 >100 mg/l	S	12	ALK 35
	Fathead minnow	96-h LC50 69 mg/l	S	22	HD 40
	Channel catfish	96-h LC50 >100 mg/l	S	22	pH 7.2
	Bluegill	96-h LC50 >100 mg/l	S	22	
Di-(2-ethyl)-hexyl-phosphoric acid[310]	Rainbow trout	96-h LC50 48.0–54.0 mg/l	S	15	HD 43 pH 8.5
Diflubenzuron (25% WP)[135]	Rainbow trout	96-h LC50 240 mg/l	S	12	ALK 35
	Fathead minnow	96-h LC50 430 mg/l	S	22	HD 40
	Channel catfish	96-h LC50 370 mg/l	S	22	pH 7.2
	Bluegill	96-h LC50 660 mg/l	S	22	
4-Dimethylamino-3,5-xylenol[130]	Bluegill	96-h LC50 7.2 mg/l	S	12	ALK 30–35 HD 40–48 pH 7.5
		96-h LC50 0.36 mg/l	S	12	HD 40–48 pH 7.9
1,1-Dimethylhydra-zine[213]	Guppy	24-h LC50 78.4 mg/l	S	22–24.5	ALK 185–232
		48-h LC50 29.9 mg/l	S	22–24.5	DO 6.9–7.8
		72-h LC50 17.2 mg/l	S	22–24.5	HD 400–500
		96-h LC50 10.1 mg/l	S	22–24.5	pH 7.8–8.2
		24-h LC50 82.0 mg/l	S	22–24.5	ALK 16–18
		48-h LC50 45.5 mg/l	S	22–24.5	DO 6.9–7.8
		72-h LC50 32.4 mg/l	S	22–24.5	HD 20–25
		96-h LC50 26.5 mg/l	S	22–24.5	pH 7.8–8.2
Dimethylnitrosa-mine[318]	Rainbow trout	10-d LD50 1 770 mg/kg	IP[1]	12	—
Dinonylnaphthalene sulfonate (50%)[310]	Rainbow trout	96-h LC50 10 mg/l	S	15	HD 43 pH 8.5

TABLE I—(*Continued*)

Pollutant	Species	Results	Exposure Type	Temp. (°C)	Test Conditions
Diquat[177]	*Rasbora trilineata*	96-h LC50 29.9 mg/l	S	—	—
	Guppy	96-h LC50 50.2 mg/l	S	—	—
Ekalaux[126]	*Saccobranchus fossilis*	96-h LC50 1.55 mg/l	S	—	—
Ekatin[126]	*Saccobranchus fossilis*	96-h LC50 11.0 mg/l	S	—	—
Endrin[148]	Flagfish	96-h LC50 0.85 μg/l	F	25	A 1.7–4.6
		110-d MATC 0.22–0.30 μg/l	F	25	ALK 39–44
		30-d BCF 9 100	F	25	HD 41–46
		65-d BCF 15 000	F	25	pH 7.3–7.6
		110-d BCF 7 100	F	25	
		30-d BCF 12 000	F	25	
Endrin (20% EC)[120]	Chingatta	96-h LC50 0.007 mg/l	S	24	DO 5.0–7.2
		96-h LC50 0.007 mg/l	S	35	HD 60–108
		96-h LC50 0.007 mg/l	S	24	pH 7.5–8.4
		96-h LC50 0.006 mg/l	S	24	
Endrin[121]	*Coregonus lavaretus*				
	Embryo	76-h LC50 3.4 μg/l	S	18	pH 6
	Larvae	72-h LC50 0.06 μg/l	S	18	
	Coregonus peled				
	Embryo	72-h LC50 766 μg/l	S	18	
	Larvae	72-h LC50 35 μg/l	S	18	
		72-h LC50 0.6 μg/l	S	18	
	Rainbow trout				
	Embryo	72-h LC50 7.7 μg/l	S	18	
		72-h LC50 7.8 μg/l	S	18	
		72-h LC50 0.6 μg/l	S	18	
	Larvae	72-h LC50 <0.25 μg/l	S	18	
		72-h LC50 0.07 μg/l	S	18	
		72-h LC50 <0.06 μg/l	S	18	
		72-h LC50 >0.12–<0.25 μg/l	S	18	
Endrin[131]	Carp				
	Embryo	72-h LC50 >256 μg/l	S	20	pH 6
		72-h LC50 49 μg/l	S	20	
	Larvae	72-h LC50 31 μg/l	S	20	
	Juvenile	72-h LC50 0.9 μg/l	S	20	
	Carassius carassius				
	Juvenile	72-h LC50 28 μg/l	S	15	
	Tench (*Tinca tinca*)				
	Juvenile	24-h LC50 2.6 μg/l	S	25	
		72-h LC50 16 μg/l	S	15	
		72-h LC50 9.1 μg/l	S	20	
	Leucaspius delineatus	72-h LC50 21 μg/l	S	15	
		72-h LC50 13 μg/l	S	20	
		72-h LC50 >2–<4 μg/l	S	25	
	Gobio gobio	24-h LC50 7.3 μg/l	S	20	
		72-h LC50 1.5 μg/l	S	15	
	Guppy	72-h LC50 0.5 μg/l	S	15	
		72-h LC50 0.3 μg/l	S	18	
		72-h LC50 0.12 μg/l	S	20	
		72-h LC50 0.08 μg/l	S	22	
		72-h LC50 0.06 μg/l	S	25	
	Stizostedion lucioperca				
	*Juvenile	24-h LC50 7.5 μg/l	S	20	
		72-h LC50 4.8 μg/l	S	15	

TABLE I—(*Continued*)

Pollutant	Species	Results	Exposure Type	Temp. (°C)	Test Conditions
9,10 Epoxystearic acid[408]	Rainbow trout Juvenile	96-h LC50 1.5 mg/l	S	—	—
Ethanol[309]	Rainbow trout	24-h LC50 11 200 mg/l	F	10	HD 90 pH 8.0
2-Ethylhexanol[310]	Rainbow trout	96-h LC50 32–37 mg/l	S	15	HD 43 pH 8.5
Fenitrothion (50% E)[138]	*Channa punctatus*	180-h LC50 1.5 mg/l	R	—	—
n-Hexadecane[331]	Atlantic salmon Eggs	168-h BCF 5.6	S	4	—
Houghtosafe 1120[323]	Rainbow trout	24-h LC50 4.2 mg/l	S	10	ALK 35
		96-h LC50 1.7 mg/ml	S	10	HD 44 pH 7.4
		96-h LC50 0.65 mg/l	F	17	ALK 237
		8-d LC50 0.59 mg/l	F	17	HD 272 pH 7.4
	Fathead minnow	24-h LC50 >90 mg/l	S	17	ALK 35
		96-h LC50 35 mg/l	S	17	HD 44 pH 7.4
		96-h LC50 17 mg/l	F	17	ALK 237
		20-h LC50 8.5 mg/l	F	17	DO 272 pH 7.4
	Channel catfish	24-h LC50 130 mg/l	S	20	ALK 35
		96-h LC50 43 mg/l	S	20	HD 44 pH 7.4
		96-h LC50 >15 mg/l	F	17	ALK 237
		30-d LC50 4.5 mg/l	F	17	HD 272 pH 7.4
	Bluegill	24-h LC50 32 mg/l	S	20	ALK 35
		96-h LC50 12 mg/l	S	20	HD 44 pH 7.4
		96-h LC50 11 mg/l	F	20	ALK 237
		17-d LC50 5 mg/l	F	20	HD 272 pH 7.4
Hyamine[148]	Bluegill	60-d BCF 33.3 (whole fish minus viscera)	F	18	DO >5.0 HD 25
		60-d BCF 80.4 (viscera)	F	18	pH 6.8
Hydrazine[313]	Guppy	24-h LC50 4.60 mg/l	S	22–24.5	ALK 185–232
		48-h LC50 3.98 mg/l	S	22–24.5	DO 6.9–7.8
		72-h LC50 3.85 mg/l	S	22–24.5	HD 400–500
		96-h LC50 3.85 mg/l	S	22–24.5	pH 7.8–8.2
		24-h LC50 3.32 mg/l	S	22–24.5	ALK 16–18
		48-h LC50 1.58 mg/l	S	22–24.5	DO 6.9–7.8
		72-h LC50 0.82 mg/l	S	22–24.5	HD 20–25
		96-h LC50 0.61 mg/l	S	22–24.5	pH 7.8–8.2
Isopimaric resin acid[408]	Coho salmon Juvenile	96-h LC50 0.22 mg/l	R	—	—
Isopimarol[408]	Rainbow trout Juvenile	96-h LC50 0.3 mg/l	S	—	—

TABLE I—(*Continued*)

Pollutant	Species	Results	Exposure Type	Temp. (°C)	Test Conditions
Jet fuel (JP-9)[325]	Golden shiner	96-h LC50 2.0 mg/l (emulsion)	R	—	HD 25
		96-h LC50 470.0 µl/l (unemulsified)	R	—	
Kathon LP[149]	Bluegill	67-d BCF 165 (whole fish minus viscera)	F	18	DO >5.0 HD 25
		67-d BCF 1 280 (viscera)	F	18	pH 6.8
LAS[307]	Rainbow trout Juvenile	96-h LC50 0.36 mg/l (untreated)	F	—	HD 250
		96-h LC50 29.5 mg/l (treated in activated sludge)	F	—	
LAS[379]	Goldfish	24-h LC50 7.59 mg/l	F	20–22.5	HD 45–96
		48-h LC50 7.48 mg/l	F	20–22.5	pH 7.1–9.3
		72-h LC50 7.00 mg/l	F	20–22.5	
		96-h LC50 6.16 mg/l	F	20–22.5	
Lead (lead nitrate)[274]	Rainbow trout	21-d LC50 2.4 mg/l	F	10.3–11.2	ALK 90 DO 7.1–10.7 HD 135 pH 7.7
Lignin sulfonate (sodium lignin sulfonate)[412]	Rainbow trout	48-h LC50 7 300 mg/l	R	9	HD 370 pH 7.3
Lignosulphonate[412]	Rainbow trout	48-h LC50 7 300 mg/l	S	9	HD 370 pH 7.31
Malathion (50% EC)[128]	Chingatta	96-h LC50 7.60 mg/l	S	24	DO 5.0–7.2
		96-h LC50 7.35 mg/l	S	35	HD 60–108
		96-h LC50 7.05 mg/l	S	24	pH 7.5–8.4
		96-h LC50 6.95 mg/l	S	24	
Malathion[148]	Flagfish	96-h LC50 349 µg/l	F	25	A 1.7–4.6
		110-d MATC 8.6–11 µg/l	F	25	ALK 39–44 HD 41–46 pH 7.3–7.6
Manganese[419]	Fathead minnow	128-h BCF 22.6	F	21–22	ALK 254 DO 7.6–8.0 HD 330 pH 8.0
MCPA[175]	Brown trout	24-h LC50 147 mg/l	S	10	—
Methylcyclohexane[325]	Golden shiner	96-h LC50 72.0 mg/l (emulsion)	R	—	HD 100
		96-h LC50 240.0 µl/l (unemulsified)	R	—	—
	Rainbow trout	42-d BCF 150	F	—	—
Methylene chloride[312]	Fathead minnow	96-h LC50 193 mg/l	F	12	HD 96
		96-h LC50 310 mg/l	S	12	pH 7.8–8.0
2-Methyl-4-isothiazolin-3-one calcium chloride[149]	Bluegill	60-d BCF 4.8 (whole fish minus viscera)	F	18	DO >5.0 HD 25
		60-d BCF 40.1 (viscera)	F	18	pH 6.8
Methylmercury[232]	Japanese medaka	96-h LC50 88 µg/l	S	—	—

TABLE I—(*Continued*)

Pollutant	Species	Results	Exposure Type	Temp. (°C)	Test Conditions
2-Methyl-naphthalene[330]	Rainbow trout	4-wk BCF (bile) 23 500	F	12	—
		4-wk BCF 40-300 (other tissues)	—	—	—
Mexacarbate[130]	Coho salmon	96-h LC50 21.0 mg/l	S	7	ALK 30–35
		96-h LC50 23 0 mg/l	S	12	HD 40–48
		96-h LC50 21.0 mg/l	S	17	pH 7.5
		96-h LC50 19.5 mg/l	S	12	ALK 10–13 HD 10–13 pH 6.6
		96-h LC50 19.7 mg/l	S	12	ALK 110–120 HD 160–180 pH 7.8
		96-h LC50 22.0 mg/l	S	12	HD 40–48 pH 6.5
		96-h LC50 4.0 mg/l	S	12	pH 9.0
	Atlantic salmon	96-h LC50 22.3 mg/l	S	12	ALK 30–35
	Brown trout	96-h LC50 20.0 mg/l	S	12	HD 40–48
	Fathead minnow	96-h LC50 23.7 mg/l	S	12	pH 7.5
Mexacarbate[130]	Bluegill	96-h LC50 22.9 mg/l	S	12	HD 40–48 pH 6.5
		96-h LC50 22.9 mg/l	S	12	ALK 30–35 HD 40–48 pH 7.5
		96-h LC50 4.3 mg/l	S	12	HD 40–48 pH 8.5
		96-h LC50 1.51 mg/l	S	12	HD 40–48 pH 9.0
		96-h LC50 0.60 mg/l	S	12	HD 40–48 pH 9.5
	Yellow perch	96-h LC50 16.2 mg/l	S	12	ALK 30–35 HD 40–48 pH 7.5
Molinate[178]	Carp	21-d LC50 0.18 mg/l	R	24	—
Monochloro-benzene[363]	Rainbow trout	24-h LD50 1.8 ml/kg	—	—	—
Monochlorodehy-droabietic acid[466]	Rainbow trout Juvenile	96-h LC50 0.6 mg/l	S	—	—
Monomethyl-hydrazine[313]	Guppy	24-h LC50 5.26 mg/l	S	22–24.5	ALK 185–232
		48-h LC50 4.02 mg/l	S	22–24.5	DO 6.9–7.8
		72-h LC50 3.67 mg/l	S	22–24.5	HD 400–500
		96-h LC50 3.26 mg/l	S	22–24.5	pH 7.8–8.2
		24-h LC50 6.89 mg/l	S	22–24.5	ALK 16–18
		48-h LC50 3.66 mg/l	S	22–24.5	DO 6.9–7.8
		72-h LC50 2.74 mg/l	S	22–24.5	HD 20–25
		96-h LC50 2.58 mg/l	S	22–24.5	pH 6.3–6.9

TABLE I—(*Continued*)

Pollutant	Species	Results	Exposure Type	Temp. (°C)	Test Conditions
MS-222[189]	Chinook salmon	5-min 50 mg/l (immobilization)	S	12	HD 100 pH 7
		30-min (lethal) 100 mg/l	S	12	
		3-h 25 mg/l (elevated cortisol)	S	12	
Naphthalene[221]	Atlantic salmon Eggs	168-h BCF 82.5	S	4	—
Naphthalene[230]	Rainbow trout	4-wk BCF (bile) 13 000	F	12	—
		4-wk BCF 40–300 (other tissues)	—	—	—
Nitrate (potassium nitrate)[97]	Guppy Fry	72-h LC50 199 mg/l	S	25–27	ALK 25.2–43.8 DO 8.30 HD 117–126 pH 7.4–7.7
Nitrate[93]	Rainbow trout				
	Fingerling (5g)	96-h LC50 0.5–10.3 mg/l	R	10	DO 8–10
	Fingerling (10g)	96-h LC50 0.9–12.1 mg/l	R	10	HD 25–300 pH 6.2–7.8
	Fingerling (5g)	96-h LC50 0.3–2.5 mg/l	R	10	DO 8–10
	Fingerling (10g)	96-h LC50 1.4–3.6 mg/l	R	10	HD 50 pH 6–8
	Fingerling (15g)	96-h LC50 0.6–1.5 mg/l	R	10	DO 8–10 HD 50 NaCl 0–200 pH 6.8 Total Chloride 1–124
Nitrite[93]	Rainbow trout				
	Fingerling (15g)	96-h LC50 0.6–31.2 mg/l	R	10	DO 8–10 $CaCl_2$ 0–200 HD 50 pH 6.8 Total Chloride 1.9–131
Nitrite[68]	Cutthroat trout Fry	96-h LC50 0.5–0.6 mg/l	F	11.5–13.3	ALK 178
		36-d LC50 0.4 mg/l	F	11.5–13.3	DO 7.3–9.0 HD 200 pH 7.72–7.98
Oil (crude 1:1 emulsion)[222]	Atlantic salmon	96-h LC50 99.0 mg/l	F	9.5–10.0	DO 10.5 pH 7.0
Palustric resin acid[400]	Rainbow trout Juvenile	96-h LC50 0.32 mg/l	S	—	—
Paraquat[177]	*Rasbora trilineata*	96-h LC50 6.99 mg/l	S	—	—
	Guppy	96-h LC50 11.5 mg/l	S	—	—
PCB: Aroclor 1248[227]	Fathead minnow	30-d LC50 4.7 μg/l	F	25	ALK 41.1
		250-d BCF 120 000	F	25	DO 7.0 HD 43.6 pH 7.52
Aroclor 1260[227]	Fathead minnow	30-d LC50 3.3 μg/l	F	25	ALK 41.1
		250-d BCF 270 000	F	25	DO 7.0 HD 43.6 pH 7.52

TABLE I—(*Continued*)

Pollutant	Species	Results	Exposure Type	Temp. (°C)	Test Conditions
PCB: Aroclor 1254[326]	Brook trout	118-d BCF 40 000–47 000	F	12	ALK 260 HD 305
		128-d 6.2 µg/l (50% mortality)	F	12	pH 7.7
Pentabromotoluene[340]	Atlantic salmon	96-h BCF (water) 23.7	S	11	—
		42-d BCF (food) 0.035	F	16	—
2,2',4,4',5-Pentachlorodiphenyl ether[340]	Atlantic salmon	96-h BCF (water) 1 414	S	11	—
		42-d BCF (food) 0.36	F	16	—
Perchloroethylene[312]	Fathead minnow	96-h LC50 18.4 mg/l	F	12	HD 96
		96-h LC50 21.4 mg/l	S	12	pH 7.8–8.0
Permethrin[132]	Rainbow trout	48-h LC50 6.0 µg/l	S	—	—
	Desert pupfish	48-h LC50 5.0 µg/l	S	—	—
	Mosquitofish	48-h LC50 97 µg/l	S	—	—
	Tilapia mossambica	48-h LC50 44 µg/l	S	—	—
Phenol[217]	Goldfish	24-h LC50 60–200 mg/l	S	5–15	DO 8.5–11
	Golden shiner	24-h LC50 35–129 mg/l	S	5–15	HD 36
	Bluegill	24-h LC50 19–160 mg/l	S	5–15	pH 7.2
	Rainbow trout	24-h LC50 5.6–11.3 mg/l	S	5–18	
Phosvel (34 EC)[124]	Chingatta	96-h LC50 3.08–31.2 mg/l	S	24–35	DO 5.0–7.2 HD 60–108 pH 7.5
Pimaric resin acid[408]	Coho salmon Juvenile	96-h LC50 0.32 mg/l	R	—	—
Pimarol[408]	Rainbow trout Juvenile	96-h LC50 0.3 mg/l	S	—	—
Pounce[123]	Mosquitofish	96-h LC50 15 µg/l	S	24	HD 100
	Channel catfish	96-h LC50 1.1 µg/l	S	24	pH 8.4
	Largemouth bass	96-h LC50 8.5 µg/l	S	24	
Primene JM-T[310]	Rainbow trout	96-h LC50 0.25–0.45 mg/l	S	15	HD 43 pH 8.5
Pydraul 50E[324]	Rainbow trout				
	Sac fry	96-h LC50 1 700 µg/l	S	—	—
	Fingerling	96-h LC50 1 330 µg/l	S	—	—
	Bluegill	96-h LC50 2 650 µg/l	S	—	—
Pydraul 50E[323]	Rainbow trout	24-h LC50 1.3 mg/l	S	10	ALK 35
		96-h LC50 0.72 mg/l	S	10	HD 44 pH 7.4
		96-h LC50 0.67 mg/l	F	17	ALK 237
		15-d LC50 0.37 mg/l	F	17	HD 272 pH 7.4
	Fathead minnow	24-h LC50 2.5 mg/l	S	20	ALK 35
		96-h LC50 1.3 mg/l	S	20	HD 44 pH 7.4
		96-h LC50 2.1 mg/l	F	17	ALK 237
		10-d LC50 1.7 mg/l	F	17	HD 272 pH 7.4
	Channel catfish	24-h LC50 7.2 mg/l	S	20	ALK 35
		96-h LC50 3.0 mg/l	S	20	HD 44 pH 7.4

TABLE I—(*Continued*)

Pollutant	Species	Results	Exposure Type	Temp. (°C)	Test Conditions
		96-h LC50 2.4 mg/l	F	17	ALK 237
		30-d LC50 2.1 mg/l	F	17	HD 272
					pH 7.4
	Bluegill	24-h LC50 4.4 mg/l	S	20	ALK 35
		96-h LC50 2.2 mg/l	S	20	HD 44
					pH 7.4
		96-h LC50 2.8 mg/l	F	20	ALK 237
		11-d LC50 2.1 mg/l	F	20	HD 272
					pH 7.4
Pydraul 115E[323]	Rainbow trout	24-h LC50 >100 mg/l	S	10	ALK 35
		96-h LC50 45.0 mg/l	S	10	HD 44
	Channel catfish	96-h LC50 >100.0 mg/l	S	20	pH 7.4
	Bluegill	96-h LC50 >100.0 mg/l	S	20	
Pyodrin[132]	Rainbow trout	48-h LC50 3.0 µg/l	S	—	—
	Desert pupfish	48-h LC50 25 µg/l	S	—	—
	Mosquitofish	48-h LC50 15 µg/l	S	—	—
	Tilapia mossambica	48-h LC50 200 µg/l	S	—	—
Rogor (30% EC)[127]	Chingatta	96-h LC50 4.48 mg/l	S	24	DO 5.0–7.2
					HD 60
					pH 7.2
Sandaracopimaric resin acid[408]	Coho salmon Juvenile	96-h LC50 0.36 mg/l	R	—	—
Silver (silver nitrate)[214]	Rainbow trout	96-h LC50 5.3 µg/l	F	9.7	ALK 28
					DO 9.7
					HD 31
					pH 6.7–6.9
		96-h LC50 6.2 µg/l	F	11.4	ALK 20
					DO 7.1
					HD 20
					pH 6.6
		96-h LC50 8.1 µg/l	F	13.5	ALK 24
					DO 7.3
					HD 26
					pH 6.7–6.8
		96-h LC50 13.0 µg/l	F	15.5	ALK 266
					DO 5.5
					HD 350
					pH 8.0
Silver (silver nitrate)[214]	Rainbow trout	18-mo MATC 0.09–0.17 µg/l	F	11.1	ALK 24.9
					DO 8.5
					HD 27.5
					pH 6.6–7.4
Sulfide[70]	Fathead minnow Juvenile	96-h LC50 6.41 µg/l	F	20.0	ALK 63–243
		96-h LC50 133 µg/l	F	20.0	DO 7.49–7.65
		96-h LC50 239 µg/l	F	20.0	HD 220
		96-h LC50 420 µg/l	F	20.0	pH 6.5–8.7
		96-h LC50 546 µg/l	F	20.0	
		96-h LC50 806 µg/l	F	20.1	

TABLE I—(*Continued*)

Poliutant	Species	Results	Exposure Type	Temp. (°C)	Test Conditions
Sulfide (hydrogen sulfide)[70]	Fathead minnow Juvenile	96-h LC50 49.2 µg/l	F	20.0	ALK 63–243
		96-h LC50 57.2 µg/l	F	20.0	DO 7.49–7.66
		96-h LC50 38.2 µg/l	F	20.0	HD 220
		96-h LC50 25.8 µg/l	F	20.0	pH 6.5–8.7
		96-h LC50 18.4 µg/l	F	20.0	
		96-h LC50 14.9 µg/l	F	20.1	
Sulfite[280]	Pugnose minnow	96-h LC50 5.68 mg/l	F	25	—
	Golden shiner	96-h LC50 4.82 mg/l	F	25	—
Sumithion[126]	*Saccobranchus fossilis*	96-h LC50 12.5 mg/l	S	—	—
Sumithion (50% EC)[127]	Chingatta	96-h LC50 12.2 mg/l	S	24	DO 5.0–7.2 HD 60 pH 7.2
TCDD[229]	Rainbow trout	105-d MATC 2.3–2 300 µg/l	F	15	—
TCDD[285]	Mosquitofish	7-d BCF 4 875	S	—	—
2,2′,4,5′-Tetrabromobiphenyl[240]	Atlantic salmon	96-h BCF (water) 474	S	11	—
		42-d BCF (food) 0.41	F	16	—
Tetrabromo-2-chlorotoluene[240]	Atlantic salmon	96-h BCF (water) 39	S	11	—
		42-d BCF (food) 0.035	F	16	—
2,3′,4,4′-Tetrachlorodiphenyl[240] ether	Atlantic salmon	96-h BCF (water) 2 720	S	11	—
		42-d BCF (food) 0.33	F	16	—
Tetrachloroguaicol[400]	Rainbow trout Juvenile	96-h LC50 0.32 mg/l	S	—	—
Tetrahydromethylcyclopentadiene[225]	Golden shiner	96-h LC50 0.51 mg/l (emulsion)	R	—	HD 100
		96-h LC50 100.0 µl/l (unemulsified)	R	—	—
	Rainbow trout	97-d BCF 9 800	F	—	—
Tetrahydronorbornadiene[225]	Golden shiner	96-h LC50 0.61 mg/l (emulsion)	R	—	HD 100
		96-h LC50 4 700 µl/l (unemulsified)	R	—	—
	Rainbow trout	97-d BCF 3 900	F	—	—
TFM[193]	Sea lamprey Larvae	96-h LC50 0.35–1.30 mg/l	S	7–12	HD 44–300 pH 6.5–8.5
	Burrowed larvae	96-h LC50 1.68 mg/l	F	12	—
	Free swimming larvae	96-h LC50 <1.48 mg/l	F	12	—
	Rainbow trout	96-h LC50 6.10 mg/l	F	12	—
	Brook trout	96-h LC50 5.95 mg/l	F	12	—
Thiodan (35% EC)[128]	Chingatta	96-h LC50 0.011 mg/l	S	24–35	DO 5.0–7.2 HD 60–108 pH 7.5–8.4
Toxaphene[140]	Brook trout Fry	150-d BCF 15 000–20 000	F	9	—
	Fathead minnow	150-d BCF 70 000–107 000	F	25	—

TABLE I—(*Continued*)

Pollutant	Species	Results	Exposure Type[a]	Temp. (°C)	Test Conditions
	Channel catfish				
	Fry	90-d BCF 40 000–91 000	F	25	—
	Adult	100-d BCF 20 000–54 000	F	25	
Toxaphene[142]	Fathead minnow	10-d LC50 4.8 µg/l	F	25	—
		259-d MATC 25–54 ng/l	F	25	—
		98-d BCF 55 000–69 000	F	25	—
	Channel catfish	9-d LC50 15 µg/l	F	—	—
		240-d MATC 129–299 ng/l	F	17–26	—
		100-d BCF 17 000–26 000	F	17–26	—
Toxaphene[141]	Brook trout Fry	15-d BCF 76 000	F	9	—
	Fathead minnow	98-d BCF 55 000–67 000	F	25	—
	Channel catfish				
	Fry	90-d BCF 27 000–50 000	—	—	—
	Adult	100-d BCF 17 000–26 000	—	—	—
2,4′,5-Tribromobiphenyl[340]	Atlantic salmon	96-h BCF (water) 1 996	S	11	—
		42-d BCF (food) 0.36	F	16	—
2,4,4′-Trichlorodiphenyl ether[340]	Atlantic salmon	96-h BCF (water) 2 298	S	11	—
		42-h BCF (food) 0.31	F	16	—
1,1,1-Trichloroethane[312]	Fathead minnow	96-h LC50 52.8 mg/l	F	12	HD 96
		96-h LC50 105 mg/l	S	12	pH 7.8–8.0
Trichloroethylene[312]	Fathead minnow	96-h LC50 40.7 mg/l	F	12	HD 96
		96-h LC50 66.8 mg/l	S	12	pH 7.8–8.0
Trichloroguaiacol[400]	Rainbow trout Juvenile	96-h LC50 0.75 mg/l	S	—	—
2,4,5-T[328]	Mosquitofish	32-d BCF 26	S	—	—
2,4,5-T[171]	Rainbow trout	96-h LC50 0.98 mg/l	R	—	—
2,4,5-T[171]	Rainbow trout	96-h LC50 8.7 mg/l (acetone solution)	R	—	—
		96-h LC50 0.15 mg/l (emulsion)	R	—	—
Tri-n-butylphosphate[310]	Rainbow trout	96-h LC50 5.0–9.0 mg/l	S	15	HD 43 pH 8.5
Tri-n-butyltin oxide[307]	Rainbow trout	24-h EC50 30.8 µg/l (Median effect concentration)	R	16	DO 7.6 HD 350 pH 7.6
	Tilapia (*Tilapia rendalli*)	24-h EC50 53.2 µg/l	R	25	DO 7.1 HD 350 pH 7.9
Tri-p-cresyl phosphate[324]	Bluegill	7-d BCF 1 589	—	—	—
Triphenyl phosphate[324]	Rainbow trout	96-h LC50 300 µg/l	S	—	—
Tris (2,3-dibromopropyl) phosphate[324]	Rainbow trout				
	Sac fry	96-h LC50 240 µg/l	S	—	—
	Fingerling	96-h LC50 1 450 µg/l	S	—	—

TABLE I—(*Continued*)

Pollutant	Species	Results	Exposure Type[a]	Temp. (°C)	Test Conditions
	Bluegill	96-h LC50 1 330 μg/l	S	—	—
n-Tritylmorpholine[335]	*Sarotherodon mossambicus* Juvenile	7-d BCF 684 55-h BCF 1 300	— R	— 27–28	— HD 291 pH 7.9–8.0
Versatic 10[210]	Rainbow trout	96-h LC50 28.0–32.0 mg/l	S	15	HD 43 pH 8.5
Zinc[217]	Goldfish	24-h LC50 24–103 mg/l	S	5–30	DO 6–11
	Golden shiner	24-h LC50 8.33–11.4 mg/l	S	5–30	HD 36
	Bluegill	24-h LC50 8.85–23.0 mg/l	S	5–30	pH 7.1
	Rainbow trout	24-h LC50 1.56–2.8 mg/l	S	5–18	
Zolone (35% EC)[127]	Chingatta	96-h LC50 0.081 mg/l	S	24	DO 5.0–7.2 HD 60 pH 7.2

[a] S = static bioassay
[b] DO = dissolved oxygen, mg/l.
[c] HD = hardness, mg/l.
[d] R = renewal bioassay.
[e] F = flowthrough bioassay.
[f] ALK = alkalinity, mg/l.
[g] BCF = bioconcentration factor.
[h] MATC = maximum acceptable toxicant concentration.
[i] A = acidity, mg/l.
[j] IP = intraperitoneal injection.

and at O_2 levels below 5 mg/l. Hamor and Garside [72] found that embryo weight in Atlantic salmon (*Salmo salar*) ova at stage 18 (advanced prehatching), and incubated in various combinations of temperature and DO, varied from 22.1 mg (5°C, 100% air saturation) to 11.9 mg (5°C, 30%) and from 14.5 mg (10°C, 100%) to 10.0 mg (10°C, 30%). Gross conversion ratios indicated that yolk was used more efficiently at 5°C (100% air saturation), but alevins become more efficient when they were in their encapsulated state, and at 10°C (100% air saturation) overtook those at 5°C before completion of yolk absorption. Kaur and Toor [73] observed that retardation of embryonic development of scale carp (*Cyprinus carpio communis*) due to low DO was less pronounced at the earlier stages than at later stages. In addition, survival and hatching rate increased directly from 4% at 1.2 mg DO/l to 98% at 12 mg/l, and the duration of incubation ranged from 120 hours at 1.2 mg/l to 68 hours at 12 mg/l. The authors concluded that this fish, economically important in India, could be successfully cultured in waters with DO levels of more than 6.0 mg/l. There was little or no effect on the occurrence of successful spawning or on the number of embryos, viability, hatching success, or survival through swim-up of black crappies exposed to constant DO concentrations of 2.5 to 6.5 mg/l, and near air saturation.[74] However, fish at 2.5 mg DO/l showed territorial behavior and started and finished spawning at earlier dates than those at higher DO concentrations. Oxygen requirements and responses to O_2 deficiency were determined by Spoor [75] for embryos and larvae of the largemouth bass (*Micropterus salmoides*). Four to nine days after fertilization, bass larvae consumed 0.021 to 0.023 μg O_2/mg·min and were most sensitive to O_2 deficiency on the sixth day. On this day, few survived exposure to 1 mg DO/l for 3 h at 20°C, and many were damaged at 2 mg/l. At 3 mg/l, the normally quiescent larvae became very active. These effects intensified with an increase in temperature. McDonald and McMahon [76] investigated the development of respiratory mechanisms in larval Artic char (*Salvelinus alpinus*) under normoxic and hypoxic conditions and found that hypoxia (pO_2 = 33 mmHg, or 25% air saturation) stimulated rhythmic ventilation within 1 hour of exposure and hatching compared to 8 to 10 days posthatching for larvae in normoxic water (pO_2 = 12 mmHg). Along with

ventilation rate, heart rate remained elevated during the 47-day exposure. Also, prolonged exposure to hypoxia retarded gill growth, although individual lamellae growth was stimulated between 38 and 47 days. Because developing embryos and sac fry of Atlantic salmon suffered a variety of adverse effects when subjected to sublethal concentrations of hydrocyanic acid, Leduc [77] concluded that to protect salmon streams during embryo-larval stages, the MATC for cyanide should not exceed 5 μg HCN/l. Hatching was delayed 6 to 9 days at 80 and 100 μg HCN/l. All concentrations (10 to 100 μg/l) reduced hatching success by 15 to 40%; conversion by yolk into body tissue during incubation was reduced at all concentrations above 10μg/l; and abnormalities ranged from 6% at 10 μg/l to 19% at 100μg/l. However, after hatching survival was unaffected and growth of cyanide-exposed sac-fry was equal to or faster than that of controls. Although juvenile bluegill sunfish (*Lepomis macrochirus*) survival was unaffected at HCN concentrations below 53.1 μg/l, fry survival was adversely affected at 15.6 to 19.4 μg/l and adult spawning was completely inhibited at 5.2 μg HCN/l (Kimball *et al.*[78]). The authors concluded that if a safe or no-adverse-effect concentration of HCN is assigned for bluegills, it must lie below 5.2 μg/l for complete life history to be accomplished in cyanide-polluted waters.

Normally defined limits for blood serum components in pond-reared channel catfish (*Ictalurus punctatus*) were compared by Warner and Williams [79] to those of catfish raised in raceways with low DO (2 mg/l at sampling). Blood urea nitrogen, total protein, uric acid, creatine, CO_2, sodium, and total bilirubin were all significantly lower in raceway-raised fish, with greatest reductions occurring in CO_2 and sodium. Alkaline phosphatase, inorganic phosphorous, calcium, cholesterol, and glucose were higher in raceway fish, but only alkaline phosphatase and glucose were significantly so. Smit and Hattingh [80] found that blood pH and pO_2 decreased; and blood pCO_2, glucose, hematocrit, and hemoglobin increased in carp (*Cyprinus carpio*) exposed to hypoxic and hypercapnic conditions, whereas hyperoxia increased only blood pO_2 and pCO_2 values. Some of these changes were more pronounced in fish with ligated pneumatic ducts. Gesser [81] observed a much higher capacity in isometrically mounted and electrically paced myocardial ventricular strips of carp to develop force during severe hypoxia and to redevelop force after it, than those of rainbow trout. When extracellular concentrations of CO_2 and HCO_3^- were raised (such that pH remained constant), the force developed during hypoxia increased, but more so in carp strips. Upon reoxygenation, contractility of carp strips were unaffected by CO_2 and HCO_3^- variations, whereas the trout strips recovered better when CO_2 and HCO_3^- levels were raised during either hypoxia or reoxygenation. Buffer capacity increased by 6 to 11 mmol/pH/l blood in young salmon exposed to high environmental carbon dioxide tension (pCO_2) for 24 hours by Börjeson.[82] After a 30-minute exposure to 25 mm Hg pCO_2, blood pH dropped 0.4 units from a control value of 7.57 and, thereafter, a continued rise in pH toward control levels paralleled a rise in plasma biocarbonate (HCO_3^-) as the fish responded to the hypercapnia-induced acidosis. Hematocrit, red blood cell count, mean corpuscle volume, and erythrocyte Cl^- content increased during the 24-hour exposure, and plasma Cl^- content was significantly depleted. A study by Eddy *et al.*[83] showed that changes in blood respiratory properties of rainbow trout kept at least 1 week in water of high CO_2 tension included elevated pCO_2, HCO_3^-, H_2CO_3 + dissolved CO_2 levels, a decrease in plasma chloride, and a drop in pH of 0.1 unit compared to control fish. Upon entering high CO_2 water, the fish suffered a sharp decrease in blood pH and significantly reduced O_2 transport by the blood, whereas trout leaving a hypercapnic environment and rapidly entering fresh water showed a sharp increase in blood pH and a decrease in blood CO_2. In either case, the fish achieved considerable compensation in response to the change in environment. Besides increases in breathing rate, breathing amplitude, heart rate, and blood pressure, Holeton [84] observed that dorsal aortic blood pH remained stable at about 8.1 when rainbow trout were exposed to water equilibrated with 5% CO and 21% O_2. The stable pH and an absence of bradycardia suggested that the observed responses were due not to acid-base related factors but rather to the stimulation of O_2 receptors, and that those O_2-sensitive respiratory control mechanisms were located at two loci.

Examination by Sousa and Meade [85] of hemoglobin absorption spectra from coho salmon exposed to elevated environmental ammonia levels revealed a progressive flattening and spectral shift similar to that observed when O_2 was stripped from the Hb molecule.

Enzymatic stimulation of glycolysis by NH_4^+ and the simultaneous suppression of TCA cycle activity caused removal of HCO_3^- from the blood to neutralize excess hydrogen ions, thereby upsetting the HCO_3^-/pCO_2 ratio. The resulting acidemia caused premature release of O_2 from hemoglobin and, if external O_2 tension was not immediately increased or ammonia stress was not diminished, the organism was susceptible to death by suffocation. Smart [86] found that increases in ventilation volume, respiratory frequency, and amplitude were associated with a 3.3-fold increase in O_2 consumption by rainbow trout exposed to an acutely lethal concentration (0.60 mg/l) of un-ionized ammonia. However, cough frequency was not significantly affected. Among blood characteristics monitored, only dorsal aortic blood pO_2 changed significantly (decreased), but heart rate doubled and dorsal aortic blood pressure increased to double the cardiac output. Sharp increases in the ammonia quotient (AQ) of the catfish (*Mystus armatus*) indicated to Sukumaran and Kutty [87] that increased protein metabolism was occurring in the fish under hypoxic conditions and during the later phases of forced exercise. The AQ, calculated from measured O_2 consumption and ammonia excretion and equal to Volume NH_3/Volume O_2, rose sharply from a normoxic value of 0.12 to 0.3 at low ambient O_2 (below 2 mg/l). The increased protein usage probably helped maintain acid-base balance and conserved sodium (Na^+) in the fish.

Pandian *et al.*[88] found that the air-breathing perch (*Anabas scandens*) increased surfacing from 550 to 650 times/day when pO_2 and CO_2 fell below 60 mm Hg or rose above 5.4 mg/l, respectively, with surfacing more dependent on pO_2 than CO_2. Observations on fed fish in aerated versus nonaerated water showed that high pO_2 elevated metabolism and increased surfacing and swimming activities, but failed to alter food utilization, suggesting that culturing *A. scandens* in aerated waters offers no special advantage. *In situ* studies by Gray and Haynes [89] showed that adult chinook salmon (*Oncorhynchus tshawtyscha*) swam deeper in supersaturated water than in normally saturated water, thus avoiding the potentially lethal conditions associated with gas bubble disease. Tagged with pressure-sensitive radio transmitters, spring 1976 fish spent 89% of their time below the critical supersaturation zone in the Snake River (Washington), while fall 1976 and spring 1977 fish, migrating in normally saturated water, were recorded significantly closer to the surface. Testing the hypothesis that changes in predator-prey interactions may be sensitive indicators of sublethal toxicity, Woltering *et al.*[90] measured food consumption and growth rates the largemouth bass predator, and observed the behavior of both the predator and its prey, the mosquitofish (*Gambusia affinis*), when both were exposed to sublethal concentrations of ammonia. Although prey consumption and growth rates of bass in control tests increased with increasing prey densities, ammonia concentrations of 0.63 and 0.36 mg/l substantially decreased prey consumption and growth of bass in tanks stocked with densities of 30, 60, and 120 mosquitofish.

pH. Wright and Snekvik [91] presented results of a 1974-1975 survey of the major ion chemistry and fish populations conducted in 700 lakes, about 250 of which were barren of fish, located in southernmost Norway. The pH gradients (4.4 to 5.2) were similar to those for sulfate concentrations while the gradient of Cl and Na concentrations were markedly steeper. The fish and chemistry data supported the well-documented observation that acid toxicity to fish is dependent on the concentrations of neutral salts. In another survey, Wright and Galloway [92] compared the acid purification and acidification of lakes occurring in northern Europe with that occurring in eastern North America. Both areas have received highly acidic precipitation (pH below 4.5) during the past 25 to 30 years. The authors found that acidification of soft and poorly-buffered fresh waters has led to a loss of fish populations. Annual pH variations due to ion separation of acid pollutants out of snow at an early melting stage and the effects on aquatic life of sudden pH-drops in Swedish lakes and streams were studied by Hultberg.[93] In caged fish studies, 53% of the seatrout (*Salmo trutta*) and 91% of the minnows (*Phoxinus phoxinus*) died when the pH range dropped to 4.30 to 4.70 in small running waters. Poor fish reproduction, *Sphagnum* sp. invasion and effects on microflora, and invertebrate deaths all corresponded to pH drops when acid substances covered littoral bottoms with a highly acid layer. Dickson,[94] who also discussed the effects of acidification of Swedish lakes, noted that acid lakes had double the acid that the pH value indicated and that Al present at levels between 150 to 600 μg/l in lakes with pH below 5 may be highly toxic to fish. Schofield [95] reported severe changes in Adirondack mountain lake and streamwater

quality due to the release of strong acid (particularly nitric acid) concentrations from snow during melting. Decreased pH and high Al concentrations in run-off were lethal to captive brook trout (*Salvelinus fontinalis*) populations. In laboratory tests, brook trout fry suffered severe gill damage when exposed to Al concentrations of 0.25 to 1.0 mg/l at pH 4.4 to 5.9, and above pH 4.0, increasing pH enhanced the toxicity of Al to trout. Dillon *et al.*[98] suggested that acidic rain either already has or will cause a severe environmental problem in south-central Ontario because precipitation samples were frequently less than pH 4.0 and because most lakes in that region have low buffering capacities (10 to 200 μeq/l).

Parsons[97] used a 1952-1954 study on the Cedar Creek Basin (Missouri) as a basis for discussing the effects of acid mine wastes on aquatic ecosystems and reviewing the physical, chemical, and biological conditions of acid lakes formed by surface mines. Although planktonic and benthic species adapted to the severe conditions in areas of Cedar Creek, which were subjected to continuous strip-mine effluent containing Fe, Cu, Pb, Zn, Al, Mg, titratable acid, and elevated pH, no fishes were observed. Fish populations were present, but variable, in downstream reaches considered unpolluted except during excessive flows of acid-mine effluents. Ryan and Harvey[98] described growth responses of rock bass (*Ambloplites rupestris*) to lake acidification due to atmospheric contamination by SO_2 in LaCloche Mountain lakes (Canada), and concluded that growth rate of fishes, in conjunction with the morphoedaphic index, may serve as an indicator of environmental stress. They found that, in lakes of greater acidity, surviving rock bass responded to decreased abundance with increased growth rate and, furthermore, that mean lake depth was the best single predictor of rock bass growth rate. Culminating 3 years of study on the effects of forest harvest on streams of the Quinault Indian Reservation (Washington), Zasoski *et al.*[99] summarized third-year findings on causes and effects of low stream pH. Most streams had pH levels between 4.3 to 5.2 and were subjected to pH levels of 3.8 to 4.2 for 3 to 4 days after autumnal rainfall. Significant correlations were found between mean autumn pH and relative biomass of all salmonids in streams, and the failure of standing stocks of salmonid fry planted in streams having low autumn pH to increase, suggested that acidic streams on the reservation have low carrying capacities. Raleigh *et al.*[100] sampled fish populations in a section of the Clinch River (Virginia) after two fish kills. The first kill occurred when a fly ash holding pond dike collapsed and dumped caustic alkaline fly ash slurry (pH 12) into the river; the second, when an undetermined amount of sulfuric acid was released into the river. A comparison of prekill, postkill, and recovery periods showed a slight decrease in number of species and relative change in abundance, but supported previous findings that fish populations can recover within a reasonably short time after a major fish kill.

In a study conducted by Coutant *et al.*,[101] acidic (pH 3.2), iron-rich seepage water from an ash pond system of a coal-fired electric power station was lethal within 24 hours to caged juvenile channel catfish (*Ictalurus punctatus*) placed in the drainage ditch and within 72 hours to fish placed near the discharge in the adjoining river. Chemical characterization of the secondary flow showed that a precipitate detrimental to fish and bottom organisms near the discharge was composed of an iron floc resulting from the acidic seepage contacting the slightly alkaline river water. Dunson *et al.*[102] compared the tolerance of three South American tetras often found in acidic blackwater rivers with that of the guppy (*Poecilia reticulata*). Whereas all guppies died after 11 days at pH 4.75, most cardinal tetras (*Cheirodon axelrodi*) could withstand immediate transfer to water at pH 3.5 for an indefinite period and with gradual adjustment, one individual survived 5 weeks at pH 3.1. A pH of 3.35 was the lower incipient lethal level (50% survival) estimated for cardinal and neon (*Hyphesobrycon innesi*) tetras. Both guppies and tetras lost large amounts of body sodium on exposure to lethal pH. Hatchery-reared brook trout, exposed to water of low pH by Swarts,[103] showed pronounced strain differences in survival ability among embryonic, juvenile, and adult trout in laboratory tests (H_2SO_4 solutions), and among juvenile trout in field tests (mine acid-polluted waters). Among other results, wild brook trout survived longer at lethal field pH than hatchery fish, larger and older fish tended to survive longer, and the rate of sodium loss at low pH was inversely correlated with resistance times. Of the factors examined in hatchery brook trout for enhancement of acid resistance, acclimation of the fish to stream or laboratory conditions of controlled pH before acid exposure and the

hereditary resistance of the given strain were most important.

Studying the survival and growth of different salmonid species to acid waters with pH > 4.4, Grande *et al.*[104] stocked the fish in small- and medium-sized acid lakes and tested them in tanks. They found that brook trout were the most tolerant to low pH followed by brown trout (*Salmo trutta*), Atlantic salmon (*Salmo salar*), and rainbow trout (*Salmo gairdneri*). Jacobsen[105] found that 18-month-old brown trout showed no significant difference in growth rate between different groups exposed for 48 days to moderately reduced pH levels of 6.26, 5.44, and 5.00 and fed a daily ration of 2.9% of their initial body weight. Jacobsen[106] also noted slower hepatic growth in brown trout yearlings exposed for 48 days to pH 5.4 and 5.0 than in fish held at a control pH of 6.3. Production of mature sperm in testes of mature flagfish (*Jordanella floridae*), exposed by Ruby *et al.*[107] to depressed pH in lake water for 20 days, was reduced by 24 to 35% at levels of 6 to 4.5, respectively, when compared with control at pH 6.7. Similarly, total volume of reproductive tissue relative to somatic tissue within the testes declined by 24 to 39%, respectively, compared to the control. An increased occurrence of empty seminiferous tubules within the testes, particularly at pH 4.5, confirmed the declining spermatogenesis. The results of an investigation by Ultsch[108] on the effect of pH on O_2 consumption in freshwater fish suggested that the importance of anoxia as a contributing factor to the death of fishes at low pH may vary among species. In fish previously exposed to pH 7.5 or 5.0 and tested in increments of 0.5 pH units over the range 4.0 to 7.0, the routine rate of O_2 consumption did not significantly decrease at pH below 5.0 for the bluegill sunfish (*Lepomis macrochirus*) or goldfish (*Carrasius auratus*), but was significantly depressed in channel catfish at pH 3.5. Breeding female brook trout clearly avoided upwellings of pH 4.0 or 4.5 in preference to ambient (pH 6.7) or alkaline (pH 8.0) upwelling, when selecting spawning sites in pH discrimination tests conducted by Johnson and Webster.[109] However, when the artificial aquifers delivered pH 5.0, ambient and alkaline discrimination was not evident. Temperature, pH, and total dissolved solids were determined by Matthews[110] as the predominant influencing factors on red shiner (*Notropis lutrensis*) habitat selection, while DO and turbidity were of limited importance. Shiners showed a strong laboratory response to pH, preferring pH 7.1 to 7.4, and in the field avoided highly alkaline conditions.

PESTICIDE POLLUTANTS

Insecticides. Vanderford and Hamelink[111] measured dieldrin, aldrin, DDT, DDE, heptachlor, and heptachlor epoxide in largemouth bass (*Micropterus salmoides*), sunfish (*Lepomis* sp.), and bullheads (*Ictalurus* sp.) from Indiana lakes and reservoirs. Only DDT, DDE, and dieldrin were detected above trace levels. The variations of pesticide levels were correlated with species of fish, season of the year, watershed land use, turbidity, and true color of the water. The occurrence of chlorinated hydrocarbons and heavy metal residues in sediments, water, and fish at American Falls Reservoir (Idaho) was reported by Johnson *et al.*[112] Chlorinated hydrocarbon pesticides did not exceed the Food and Drug Administration (FDA) recommended standard of 5 mg DDT/kg or 0.3 mg dieldrin/kg. The occurences of pesticide residues in the biota from the Upper Snake River following the collapse of the Teton Dam was measured by Perry.[113] Mean total DDT levels in the rainbow trout (*Salmo gairdneri*) were about 200 μg/kg (wet weight). Mean levels for Rocky Mountain whitefish (*Prosopium williamsoni*) and the Utah sucker (*Catostomus ardens*) varied from 139 to 601 μg/kg and 221 to 1 224 μg/kg, respectively. The range of dieldrin for these three species varied from 3 to 96 μg/kg. Five species of fish from Lake Superior (Canada) and twelve species from Lake Huron (Canada) were measured for organochlorine pesticides and PCBs between 1968 and 1975 by Frank *et al.*[114] None of the five species from Lake Superior contained residues of DDT, dieldrin, and PCBs above tolerance levels of 5, 0.3, and 2 mg/kg, respectively. Some species caught in 1975 contained trace levels of cis- and trans-chlordane. Oxychlordane, endrin, and heptachlor epoxide were not detected. Three species of fish from Lake Huron contained mean residues of DDT and PCBs above the tolerance level. Individual fish of three species contained residues above the tolerance level. Chlordane, heptachlor epoxide, and HCB were detected in some fish. Residues of organochlorine insecticides and PCBs in fish from Lakes Saint Clair and Erie (Canada) from 1968 to 1976 were also analyzed by Frank *et al.*[115] Residues of DDT peaked at 1.19 mg/kg in longnose gar (*Lepisosteus osseus*) in Lake St. Clair in

1970-1971 but declined in all species by 1975-1976. White bass (*Morone chrysops*) caught in Lake Erie had the highest residues of dieldrin at 0.17 mg/kg in 1975. PCB residues exceeded the 2.0 mg/kg tolerance level of Health and Welfare Canada in several species with only one species exceeding the 5.0 mg/kg tolerance level of the United States. Residues of DDT, mirex, dieldrin, chlordane, and PCBs were measured in Great Lakes coho salmon (*Oncorhynchus kisutch*) and chinook salmon (*Oncorhynchus tshawytscha*) by Moccia *et al.*[116] to determine if goiter frequency in these salmon correlated with organochlorine residues. PCBs and DDE represented the major organochlorine contaminants of the ones measured, accounting for 86% of the total organochlorine levels. No cause-effect relationship was seen between tissue organochlorine burdens and overt goiter frequency in Great Lakes coho salmon, and no evidence of overt goiters was observed in chinook salmon, although high organochlorine levels were present in this species. Concentrations of dieldrin, DDT, PCBs, and Hg in the longnose sucker (*Catostomus catostomus*), white sucker (*Catostomus commersoni*), and golden redhorse (*Moxostoma erythrurum*) from the upper Great Lakes were measured by Zabik *et al.*[117] Dieldrin concentrations varied from < 0.001 to 0.23 mg/kg in deboned and whole ground samples, and DDT varied from 0.005 to 0.30 mg/kg. Sackmauerova *et al.*[118] measured BHC isomers, DDE, DDT, and HCB in the water and fish from the Danube River in Czechoslovakia. The mean level of these compounds in fish were 1 000 to 10 000 times higher than in the water. Higher concentrations of DDT and DDE were found in fish as compared with BHC levels. Organochlorine pesticide residues in various organisms from different aquatic ecosystems in Iran were investigated in 1974 by Sodergren *et al.*[119] Fish obtained from Shahpour and Kupor Rivers contained appreciable amounts of DDT, up to 614 mg/kg (fat weight). Fish obtained from one lake and two reservoirs contained low to moderate levels of DDT. The low level of DDT in pike (*Esox lucius*) (up to 1.3 mg/kg) collected from the Bandar-Pahlovi Mordab suggested that low levels of organochlorine insecticides enters the food chain in this region. Analyses were made by Huschenbeth[120] to determine residues of organochlorine pesticides in marine and freshwater fishes from the North-Atlantic, North-Sea, Baltic, and Black Sea. The residues in eels were higher than in marine fish. The most contaminated eels came from Turkey and the least contaminated were from Ireland. The occurrence of organochlorine pesticide residues from fish from two South African lakes was measured by Greichus *et al.*[121] The insecticide residues most commonly found were DDE, DDD, DDT, and dieldrin. The average total concentration of these compounds was 0.32 mg/kg and 2.0 mg/kg for bluegills (*Lepomis macrochirus*) and bass (species not given), respectively, from the Vöelvlei Dam ecosystem. Two fish, canary kurper and blue kurper, from the Hartbeespoort Dam ecosystem had a total average concentration of 1.0 mg/kg and 0.97 mg/kg, respectively.

The effects of diflubenzuron on nontarget aquatic populations in a Louisiana intermediate marsh was investigated by Farlow *et al.*[122] Six applications of 28 g AI/ha over an 18-mo period caused statistically significant differences in population density of treated and untreated populations. Populations of five taxa were reduced while 15 taxa, including flagfish (*Jordanella floridae*) and mosquitofish (*Gambusia affinis*) were increased. The remaining 27 taxa were not different. Outdoor experimental streams were used by Merna and Queener[123] to study the effects on stream organisms of a simulated treatment of methoxychlor for control of black fly. Survival of yellow perch (*Perca flavescens*) was less in the two streams treated at 600 μg/l than in the streams treated at 200 μg/l or the control streams. Stauffer[124] studied the mortality of eggs and fry of lake trout (*Salvelinus namaycush*) using eggs with a high content of DDT and PCBs from Lake Michigan, and eggs with a low content of DDT and PCBs from the hatchery at Marquette, Mich. The data indicated that DDT and PCBs were not the cause of reproductive failure of lake trout in Lake Michigan.

Huner[125] measured the diurnal changes in the sensitivity of the mosquitofish to endrin. In an outdoor exposure of fish to a concentration of 1 000 μg/l, the time of immobilization decreased from dawn to dusk. In an indoor exposure, immobilization times were similar. The decrease in time of immobilization in the outdoor exposure appeared to be a function of increased temperature. The acute toxicity of chlordane, Ekalaux, Ekatin, and Sumithion to the teleost fish, *Saccbranchus fossilis*, was reported by Verma *et al.*[126] The 96-hour LC50 values for these compounds were 0.42, 1.55, 11.0, and 12.5 mg/l, respectively. Another study by Verma *et al.*[127] determined the acute

toxicity of four organophosphorus biocides to *Channa gaucha*. The 96-hour LC50 values ranged from 0.08 mg/l for zolone to 217 mg/l for abate (Table I). Toxicity was increased by an increase in temperature and pH and by a lowering of hardness. The acute toxicity of endrin, thiodan, malathion, and phosvel to *Channa gaucha* was determined by Dalela *et al.*[128] The 96-hour LC50 values ranged from 0.006 mg/l for endrin to 33 mg/l for phosvel. The effects of temperature, pH, and size of fish were investigated. The effect on survival time of pre-exposure to phenobarbitol and hexobarbital of *Nemacheilus botia*, *Nemacheilus denisoni*, *Poecilia granulosa*, and *Lepidocephalichthys thermalis*, when exposed to endrin was measured by Kachole *et al.*[129] Pre-exposure to either barbitol increased the survival time of all four species of fishes. The effects of water hardness, pH, temperature, and aging (deactivation) on the toxicity of mexacarbate to coho salmon, Atlantic salmon (*Salmo salar*), brown trout (*Salmo trutta*), fathead minnows (*Pimephales promelas*), bluegills, and yellow perch were studied by Mauck *et al.*[130] Identification and toxicity of the breakdown products of mexacarbate were also investigated. Water temperature and hardness did not affect the biological activity of mexacarbate, while pH did. The insecticide was 38 times more lethal to bluegills at pH 9.5 than at pH 6.5. Wohlgemuth [131] conducted static tests to evaluate the effects of temperature and life history stage on the acute toxicity of endrin to nine species of fishes. The 72-hour LC50 values varied from <0.06 μg/l for 29-day-old larvae of rainbow trout to 766 μg/l for 1-day-old larvae of *Coregonus peled*. Endrin was more toxic at higher temperatures. Eleuter embryos were more resistant than other developmental stages. The lethal toxicity of six new synthetic pyrethroids to mosquito larvae and four species of fish was determined by Mulla *et al.*[132] Decamethrin was the most toxic with 48-hour LC50 values ranging from 0.5 μg/l for rainbow trout to 1.0 μg/l for mosquitofish. The least toxic was ABG-6070 with 48-hour LC50 values of 225 μg/l for rainbow trout, > 1 500 μg/l for mosquitofish and desert pupfish (*Cyprinodon macularius*) and > 3 000 μg/l for *Tilapia mossambica*. The acute toxicity of Pounce, a synthetic pyrethroid, to three species of fishes was determined by Jolly *et al.*[133] The channel catfish (*Ictalurus punctatus*) was the most sensitive with a 96-hour LC50 value of 1.1 μg/l. Largemouth bass and mosquitofish were more resistant with 96-hour LC50 values of 8.5 μg/l and 15 μg/l, respectively. The acute toxicity of Altosid and Dimilin to coho salmon and rainbow trout was determined by McKague and Pridmore.[134] Dimilin was not lethal at concentrations up to 150 mg/l. The 96-hour LC50 values for Altosid were 86 mg/l for salmon and 106 mg/l for trout. The acute toxicity of diflubenzuron and four of its degradation products to three species of aquatic invertebrates and four species of fishes was determined by Julin and Sanders.[135] The 96-hour LC50 values of the (W.P.) formulation to fish varied from 240 μg/l for rainbow trout to 660 μg/l for bluegills. The most toxic degradation product was 4-chloroaniline. A 30-day exposure of eyed eggs and fingerlings to diflubenzuron (technical grade) had no observable effect at 0.30 mg/l.

The effects of DDT, BHC, and PCBs on the growth of fish were measured by Takeda.[136] Neither sublethal concentrations of DDT or sublethal doses of DDT in food inhibited growth of carp (*Cyprinus carpio*). Both sublethal concentrations and sublethal doses of BHC in food decreased food conversion efficiency and inhibited growth of carp in the first 2 weeks of exposure. However, the food conversion efficiency was recovered after the first 2 weeks and growth of exposed fish was similar to controls. Mohamed *et al.*[137] used a respirometer to study the effects of sublethal exposures of DDT on the metabolism of two carps, *Cirrhina mrigala* and *Labeo rohita*, and the giant gourami, *Colisa fasciata*. The metabolism of all three species increased on exposure of 0.02 DDT/l and the increase was greater with increased exposure times. Saxena and Garg [138] conducted studies to determine the effect of fenitrothion and carbaryl on the maturation of oocytes in *Channa punctatus*. The fish were exposed for 150 days to 180-day LC50 concentrations of 1.5 mg/l of fenitrothion (50% E. C.) and 2.0 mg/l of carbaryl (50% W. P.). The results suggested that both insecticide exposures arrested ovarian development. After 150 days, there were no mature oocytes in fish exposed to the insecticides while in control fish about 50% of the oocytes were mature. Ovary weight in the exposed fish was only about 50% of the control. The effects of dietary fenitrothion on the growth and hierarchical position of brook trout (*Salvelinus fontinalis*) was studied by Wildish and Lister.[139] At a dietary level of 10 mg/kg, individual fish received doses of fenitrothion that varied from 13 to 136 ng/kg·d. At these

doses the insecticide did not affect growth, food conversion efficiency, or hierarchical position.

Mehrle and Mayer [140, 141] summarized studies on the long-term exposure of fish to toxaphene. Fry were more sensitive than adult fish. At all experimental concentrations, including the low concentration of 39 ng/l, there was a significant effect on bone composition and growth of fry of the brook trout exposed for 90 days. The weight of fathead minnow fry exposed for 30 days was reduced at 54 ng/l but not at 25 ng/l. Channel catfish fry exposed for 90 days had reduced survival and growth at 299 ng/l, and collagen composition was reduced at 72 ng/l. The amount of toxaphene in the fish varied from 15 000 to 10 700 times the water concentration. The acute and chronic toxicity of toxaphene to the fathead minnow and channel catfish was also determined.[142] The LTC values were 4.8 μg/l (10-day LC50) for the minnow and 15 μg/l (9-day LC50) for the catfish. The lowest concentrations having a significant effect ($P = 0.05$) on survival, growth, and reproduction were 54 ng/l for the fathead minnow and 299 ng/l for the channel catfish. The effects on collagen and hydroxyproline in channel catfish were significant at lower toxaphene concentrations than the effect on growth. The maximum toxaphene accumulation from water to fish was 69 000 times for the fathead minnow and 50 000 times for the channel catfish. Brozio and Litzbarski [143] examined the effect of toxaphene on the blood profile, enzymes, liver, and gills of carp. The interaction of toxaphene and vitamin C was studied by Mayer *et al.*[144] Three groups of channel catfish, fed diets containing 63, 670, or 5 000 mg/kg of vitamin C, were exposed to different concentrations of toxaphene for 150 days. Diets containing 670 or 5 000 mg/kg of vitamin C increased the tolerance of fish in most toxaphene concentrations and reduced whole body residues. On the basis of this study, the researchers suggested that the dietary vitamin C concentrations should be greater than the recommended 100 mg/kg for hatchery-reared or cage-cultured catfish. Auwarter [145] used a flow-through exposure system to study interactions of toxaphene and methyl parathion on toxicity and accumulation in bluegills. The effect of mixtures on short-term survival was antagonistic and all short-term interactions that were significant for pesticide accumulation were antagonistic. Long-term studies revealed a synergistic action on accumulation of both pesticides. The acute and chronic toxicity of diazinon to fathead minnows and brook trout was studied by Allison and Hermanutz.[146] Fathead minnows exposed to the lowest concentration (3.2 μg/l) from 5 days after hatch through spawning had a significantly ($P = 0.05$) higher incidence of scoliosis than the controls, and hatchability of their progeny was reduced by 30%. Exposure of mature brook trout for 6 to 8 months to the low test concentration of 0.55 μg/l resulted in reduced growth rates for their progeny. Average 96-hour LC50 values for diazinon were 7.8, 0.77, 1.6, and 0.46 mg/l, respectively, for fathead minnows, brook trout, flagfish, and bluegills. These data indicated application factors (MATC/96-hour LC50) in the order of 10^{-4} or lower. Concentrations of diazinon in muscle tissue of brook trout exposed for 8 months varied from 19 to 51 times the water concentration. Allison [147] investigated the relationship of exposure duration, concentration, and periodicity to the toxicity of diazinon to flagfish. The fish were exposed throughout one and a half generations to establish baseline chronic toxicity. Three additional tests were made with concentrations increased and duration decreased 4-fold. These exposures were made as a single pulse to newly hatched, prespawning and spawning fish. All four exposures had similar overall effects on growth and survival at equivalent exposure units. This study indicated that exposure units can be used to assess the effect of fluctuating water concentrations of organophosphate pesticides over a wide range of concentration, duration, and periodicity. The acute and chronic toxicity of endrin and malathion to flagfish was determined by Hermanutz.[148] The 96-hour LC50 for endrin was 0.85 μg/l and the lowest concentration having a significant effect in the chronic study was 0.30 μg/l. Mean accumulation factors for various life stages and exposure periods varied from 7 100 to 15 000. The 96-hour LC50 for malathion was 349 μg/l and the lowest concentration having a significant effect in the chronic exposure was 11 μg/l.

Krzeminski *et al.*[149] used the compartmental analysis approach of pharmacokinetics to develop a simple mathematical model to predict, by computer simulation, steady-state pesticide residues in fish from pre-steady-state data. The model was applied to data from exposure of bluegills to four different biocides. The values predicted from the model were in good agreement with experimental values. The uptake,

distribution, and elimination of chlordane by goldfish (*Carassius auratus*) was measured by Moore *et al.*[150] Goldfish exposed for 3 days to 3.4 μg/l of a 1:1 mixture of ^{14}C-alpha- and gamma-chlordane had a bioconcentration factor of 162. After transfer to fresh water, about 90% of the chlordane was eliminated in 11 days. Feeding chlordane contaminated daphnids to goldfish resulted in residue levels of 0.05 and 0.12 mg/kg after 1 and 7 days, respectively. The effect of size on the accumulation and elimination of dieldrin in muscle tissue of channel catfish exposed to dieldrin in water, food, and both water and food was studied by Shannon.[151] After 28 days of exposure, large fish accumulated higher levels of dieldrin than did small fish, but after 20 days of elimination, dieldrin levels were similar in both size groups. Large fish exposed for 29 days to dieldrin concentrations of 75 μg/l in water, 2 mg dieldrin/kg food in the diet, and to the same amounts in both water and food contained dieldrin in muscle tissue of 274, 1 243, and 2 418 μg/kg, respectively. The absorption, accumulation, and elimination of pesticides by several species of fish and invertebrates in a model ecosystem was studied by Matsumura.[152] Various factors such as the amount of pesticides, size of ecosystem, temperature, and physical characteristics of the the pesticides were studied in relation to the rate of pesticide uptake and elimination by the test organisms. The design of the model was also evaluated. Booth and Ferrell [153] studied the degradation and accumulation properties of Dimilin in a model ecosystem. Following multiple application, Dimilin disappeared from water, sediment, and aquatic vegetation after several days. After a 28-d test period, channel catfish did not bioaccumulate Dimilin residues from treated soil. Tests were conducted by Granett *et al.*[154] to determine whether Dimilin-Gl, a candidate larvicide, produced avoidance reactions in male Atlantic salmon parr at larvicidal concentrations. Salmon appeared to be repelled by Dimilin-Gl and the Florex carrier at concentrations at or below projected practical treatment levels for black fly control. Because the repellency of Dimilin-Gl was no more than that of the Florex alone, however, the carrier apparently was more important to the behavioral response than was the insecticide.

Addison and Willis [155] examined the metabolism by rainbow trout of p,p′[^{14}C] DDT and nine related compounds proposed or demonstrated to be products or intermediates in the degradation of p,p′DDT by vertebrates. The results indicated that rainbow trout have no elaborate system for the metabolic degradation of p,p′DDT. Addison *et al.*[156] fed p,p′DDT and p,p′DDE to brook trout for 4 weeks and produced tissue concentrations of about 20 and 60 mg/kg (wet weight), respectively. This exposure did not induce the activities of the mixed function oxidase enzymes of aniline hydroxylase, nor of microsomal protein levels above controls. A continuous steady-state assay procedure was used by Jackson and Gardner [157] to investigate the *in vitro* effect of DDT and several analogs on the ATPases of brain of brook trout. The results suggested that the *in vitro* inhibition of mitochondrial ATPase is of questionable significance in regard to the symptoms of acute DDT poisoning. Jackson and Gardner [158] also investigated the *in vitro* effects of temperature, DDT and several analogs on trout brain MG^{2+}-ATPases specific activity. It was suggested that DDT has the potential to allosterically affect membrane-bound enzymes by becoming a major lipoid component of the membranes. Coleman *et al.*[159] studied the effect of p,p′DDT on the ultrastructure of mitochondria-rich "chloride" cells in the gills of *Tilapia aurea* kept in fresh and sea water. The ultrastructure of "chloride" cells of fish injected with DDT was not different than the control fish. The effect of DDT on the lateral line nerve of steelhead trout was examined by Peters and Weber.[160] It was found that exposure of DDT did not change the spontaneous activity of nonbursting fibers or alter the stimulus threshold. The effect of DDT on repetitive activity was temperature dependent and occurred when temperature was lowered. The α-amylase activity of liver of *Anabus testudineus* exposed to endrin was examined by Kumar and Bhattacharya.[161] Hepatic α-amylase activity of exposed fish was inhibited. *In vitro* studies indicated that inhibition was concentration dependent. The inhibition of α-amylase activity was reversed by returning endrin-exposed fish to control water containing added sodium chloride. The histopathological effects of sublethal concentrations of diazinon, methyl parathion, and dimethoate on the liver of *Channa punctatus* were examined by Anees.[162] All three organophosphate insecticides produced detectable liver pathology. Mukhopadhyay and Dehadrai [163] investigated the effects on the microsomal drug metabolizing enzymes from liver and gills of the air-breathing catfish (*Clarias bactrachus*) exposed to 1 mg/l of

malathion for 30 days. Fish exposed to malathion had a significant increase in N- and O-demethylase and increase in cytochrome P-450 content of both liver and gill tissue. Whitemore and Hodges [164] investigated the *in vitro* inhibition of muscle esterases of the mosquitofish by several pesticides. They found that the O_2 analogs of several organophosphates were potent inhibitors of muscle esterases. Malathion did not inhibit esterases unless preincubated with muscle extracts before electrophoresis. In a preliminary study, Madder and Lockhart [165] measured the effects of two insect growth regulators on five blood serum parameters of rainbow trout. Diflubenzuron produced a significant decrease in GOT activity, and methoprene caused a significant decrease in glucose values. Buangsawasdi [166] investigated the toxicity of organophosphate insecticides to rainbow trout with respect to temperature and site of action. The results indicated that the cardiovascular and respiratory systems were important sites of action and that toxicity was related to physio-chemical properties of the pesticides. The effect of acclimation temperature, age, and malathion concentration on the temperature selection response of the common shiner (*Notropis cornutus*) was studied by Domanik and Zar.[167] All three factors affected the temperature selection response of fish exposed to malathion.

Herbicides. The distribution and degradation of six dinitroaniline herbicides in a laboratory ecosystem were investigated by Kearney *et al.*[168] The bioconcentration factors (based on ^{14}C) for the mosquitofish (*Gambusia affinis*) varied from 32 for fluchloralin to 83 for dinitramine. Fish from aquariums held in the dark had bioconcentration factors that varied from 253 for dinitramine to 755 for butralin. Fish exposed for 12 days to 10 μg/l of profluralin test solution, renewed at 4-day intervals, had a rapid increase in bioaccumulation factors up to about 1 200 for 4 days and then a decrease. Channel catfish (*Ictalurus punctatus*) exposed to the same concentration had a continuous increase for 20 days when the bioaccumulation ratio was 11 000. The distribution of oxadiazon and phosalone in a model ecosystem containing algae, snails, daphnids, and fish was measured by Ambrosi *et al.*[169] The organisms accumulated oxadiazon and phosalone 30 to 300 times greater than the water concentration, with mosquitofish having the higher accumulation. Seven degradation products of ovadiazon were found. Shadoff *et al.*[170] conducted a search for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), an impurity found in 2,4,5,-T, in two locations in Texas where it would most likely accumulate from the approved domestic use of 2,4,5-T. With a detection limit of less than 10 ng/l, TCDD was not found in catfish, bass, and walleye (*Stizostedion vitreum vitreum*).

The acute and subacute toxicity of 2,4,5-T to rainbow trout (*Salmo gairdneri*) was assessed by Yokote *et al.*[171] The authors reported that LC50 values were lower when the herbicide was applied as an emulsion than when applied as an acetone solution. In the subacute studies, trout were exposed for 12 weeks under flow-through conditions. Weedon, a commercially available emulsion formulation of 2,4,5-T buthoxyethanol ester, at a concentration of 0.20 mg/l retarded the growth of fish and caused pathological changes in the liver, kidney, heart, and skin. Reagent grade 2,4,5-T (emulsified with Pegnol-1000) at a concentration of 0.50 mg/l had no adverse effect on growth, but caused slight pathological changes in the same tissues. Fish exposed to Pegnol-1000 at a concentration of 0.425 mg/l showed no adverse effect. Chambers *et al.*[172] found that mosquitofish from an insecticide resistant population had higher 48-hour LC50 values to two thiol esters of 2,4-D than did fish from a susceptible population. Fish from both populations exposed to 0.1 mg/l of DEF for 24 hours before exposure to the esters were more resistant than nonexposed fish. Esterase activity in gill and liver preparations showed that the resistant fish had lower levels of liver esterase than the susceptible fish, but results with gill preparations were not consistent. DEF inhibited activity in both tissues from both populations. Stalling and Huckins [173] exposed bluegills (*Lepomis macrochirus*) in outdoor pools to one 2 mg/l application of ^{14}C labeled dimethylamine salts of 2,4-D. They found that ^{14}C was incorporated into both free fatty acids and the fatty acid moiety of triglycerides from fillets and head-viscera samples. In addition ^{14}C was detected in glycogen from fish liver extracts. The authors suggested that a significant amount of the aromatic moiety of DMA-^{14}C-2,4-D was completely degraded, probably by microorganisms and other factors within the pool, and that these ^{14}C fragments were then metabolically incorporated by the bluegills. The uptake and metabolism of DMA-[^{14}C]-2,4-D by bluegills and channel catfish was studied by Sikka *et al.*[174] In a 7-day exposure to 2 mg/l, the maximum concentration was reached within

24 hours with a total body concentration of 0.93 mg/kg for bluegill and 0.20 mg/kg for channel catfish. Most of the radioactivity absorbed by the fish was in the head and viscera. The results indicated that bluegills and channel catfish did not metabolize 2,4-D. Hattula *et al.*[175] studied the toxicity of the sodium salt of 4-chloro-2-phenoxyacetic acid (MCPA) to two species of fish. The 24-hour LC50 was 147 mg/l for the brown trout (*Salmo trutta*). This species was exposed for 28 days to sublethal concentrations of 10 and 30 mg/l. Histopathological analysis did not show any change in the tissues. Chemical analysis of the exposed fish gave concentrations of 3 to 4 mg/kg (wet weight) of MCPA and 1 to 2 mg/kg (wet weight) of 4-chloro-o-cresol. Goldfish (*Carassius auratus*) exposed for 42 days to 40 and 60 mg/l did not show any histopathological effects when compared to the controls. The toxicity of molinate to carp (*Cyprinus carpio*) was investigated by Kawatsu.[176] The 21-day LC50 value was 0.18 mg/l. Anemia was found in fish exposed to this herbicide, with microaneurysm and punctata hemorrhage being observed in gills of anemic carp. Histopathological effects were swelling of gill lamella, dilation and PAS-positive thickening of capillar wall, and deterioration of pillar cells. Laboratory tests to screen for herbicides used for the control of an aquatic plant, *Salvinia molesta*, were performed by Kam-Wing and Furtado.[177] The tests included the toxicity effect of herbicides to two species of fish and one aquatic invertebrate. The 96-hour LC50 values for fish ranged from 1.59 mg/l for Tok E-25 to 222.8 mg oakaphon/l for the guppy (*Poecilia reticulata*) and from 0.756 mg/l for Tok E-25 to 136.2 mg dalaphon/l for *Rasboro trilineatus*. Olson *et al.*[178] exposed carp to 1 mg/l of dinitramine for 12 hours. Concentrations of dinitramine and metabolites were measured at 0, 1, 3, and 7 days past exposure. After the fish were held in fresh water for 7 days, samples of plasma, muscle, and gallbladder contained 6, 25, and 7%, respectively, of concentrations found immeditely after exposure. Metabolites were not found in plasma or muscle but deethylated dinitramine was present in gallbladder bile indicating biotransformation by dealkylation. Ishikawa *et al.*[179] studied the effects of the herbicide, sodium pentachlorophenolate (PCP-Na), and the fish anesthetic, 2-amino-4 phenylthiazole (APT), on the enzyme activities of lysosomal origin in the hepatopancreas of carp. Changes in relative specific enzyme activities of β-glucuronidase, acid phosphatase, and acid ribonuclease were observed with both PCP-Na and APT. Comparison of the changes in the activities of the three enzymes during culture in a medium containing 0.1 mg/l PCP-Na and those in a medium containing 10 mg/l APT showed significantly different patterns, suggesting the mechanism of effects may be different from each other. Fish exposed to combined herbicidal preparations (Kerpopur, Bandex, Dikogren) containing 2-methyl-4-chlorphenoxyacetic acid were analyzed for histopathological effects by Groch and Svobodova.[180] Both carp and rainbow trout had circulatory disorders. The occurrence of skin and gill lesions on channel catfish exposed to 0.5 mg/l nifurpirinol (Furanace, P-7138) was reported by Mitchell *et al.*[181] Skin lesions first appeared 3 days after the end of the 4-day exposure to Furanace, and by the twelfth day 63% of the test fish had visible lesions; while in the 14-day test, lesions appeared on the eleventh day, and 41% of the fish had visible lesions by the end of the test. Lesions continued to develop, new lesions formed, and mortalities occurred after the test fish were moved to untreated water. Gill lesions that occurred in fish exposed to Furanace were characterized by hyperplasia, hypertrophy, oedema, and shortening of the lamellae.

Piscicides and Anesthetics. The uptake, distribution, and elimination of a 1:8 mixture of ^{14}C Bayer 2353 and Bayer 73 by largemouth bass (*Micropterus salmoides*) was reported by Schultz and Harman.[182] Radioactivity was found in all tissues and organs, with the concentration being highest in the gallbladder and lowest in the muscle. In the elimination study, ^{14}C material decreased with time in all organs and tissues except for the gallbladder bile. Dawson *et al.*[183] studied the effect of water quality on the acute toxicity of TFM, Bayer 73, and a 98:2 mixture of the two tested against the larvae of sea lamprey (*Petromyzon marinus*). Temperature and hardness had little influence on toxicity, but toxicity was increased in water of low pH. The effect of the mixture of the two piscicides was evaluated by the use of an additive index. Rehulka [184] examined the toxicity of malachite green B to carp (*Cyprinus carpio*). It was found that concentrations for disease treatment should vary with the age and health of the fish. The maximum concentration for a 6-day exposure should not exceed 0.3 mg/l. The effectiveness of injecting sockeye salmon (*Oncorhynchus*

nerka) with a mixture of antibiotics containing 50% penicillin-streptomycin and 50% oxytetracycline for preventnig prespawning mortality was evaluated by Williams and Stelter.[185] Administration of the combiotic promoted healing of gill lesions among 33 to 77% of the fish arriving with lesions, compared with only 4.5 to 13.3% healing in the controls. Spawning success was increased from 0% in the control fish to 50 to 85.7% in treated fish. It appeared that spawning success or sockeye populations could be enhanced with administration of a combiotic; however, the procedure used would not be practical for use on entire populations. Ohi [186] reported that an intracranial dose of 2 μg of actinomycin D inhibited about 70% of brain RNA synthesis in the goldfish. It was suggested that brain RNA synthesis was necessary for the formation of long-term memory but not for short-term memory. Residue concentrations of the antioxidant, ethoxyquin (EMQ) in tissues of Atlantic salmon (*Salmo salar*) were investigated by Skaar and Roald.[187] Small fish maintained in fresh water and fed 900 mg EQM/kg for 2 months had 0.7 mg/kg of EMQ and possible metabolites in muscle tissue. The half-life in muscle was between 4 to 6 days. The fish, fed 150 mg EMQ/kg in their diet for 2 months, had residue levels of EMQ and possible metabolites of 0.3 mg/kg in blood, 0.7 mg/kg in muscle, 0.8 mg/kg in kidney, and 1.8 mg/kg in hepatic tissue.

Reinitz and Rix [188] found that exposing rainbow trout (*Salmo gairdneri*) to 70 mg/l of tricaine methanesulfonate (MS-222) resulted in a linear increase in hematocrit values between a 3- to 9-minute exposure. Handling of unanesthetized trout caused a higher and more variable hematocrit value than an exposure to MS-222 of up to 3 minutes. Strange and Schreck [189] found that a brief exposure to 50 mg/l of MS-222 immobilized chinook salmon (*Oncorhynchus tshawytsha*) as rapidly as a lethal concentration of 100 mg/l and that it was less stressful in terms of the cortisol stress response than 25 mg/l, which merely depressed fish activity. Oswald [190] evaluated anesthetic drugs by intramuscular or intraperitoneal injection in rainbow trout. Saffan gave superior long-duration anesthesia, and Epontol was the best short-duration drug. Investigations on the effect of temperature and hydrostatic pressure on recovery time of rainbow trout from pentobarbital anesthesia were made by Belaud *et al.*[191] The data showed a similarity between the effects of temperature and pressure on the recovery and on chemical kinetics. Fish anesthetized with pentobarbital at 500 mg/l for 15 minutes had a recovery time that varied from 10 minutes at 20°C to 82 minutes at 10°C. Booke *et al.*[192] evaluated sodium bicarbonate for field use as a fish anesthetic using rainbow trout, brook trout (*Salvelinus fontinalis*), and carp. A combination of pH 6.5 and 642 mg/l of sodium bicarbonate was the most effective treatment for causing fish, within 5 minutes, to cease locomotion and slow opercular rate but to retain reflex response to pressure on the caudal fin. Spangenberg [193] discussed the suitability of trichlorobutylalcohol as a narcotic for *Ctenopharynogodon idella* and rainbow trout. Complete narcosis occurred in water temperatures and trichlorobutylalcohol concentrations of 15°C and 450 mg/l, 20°C and 400 mg/l, 25°C and 300 mg/l, 30°C and 150 to 200 mg/l.

CHEMICAL POLLUTANTS

Inorganics. Davis [194] analyzed 18 species of freshwater fish from lentic waters to determine if concentrations of elements in water significantly affect their composition. There was no significant relationship between Ca, Mg, Na, K, N, S, P, Zn, and Cu concentrations in fish and the waters from which they were taken. Analysis of two size series of bluegills (*Lepomis macrochirus*) indicated positive correlation with length and concentrations of Ca, Na, P, and Mn. Negative correlations were shown for N, K, S, and Zn. Several Colorado and Wyoming lakes, some of which receive seleniferous seepage from cretaceous shales, were sampled by Birkner [195] in order to investigate the environmental cycling of Se. In lakes containing less than 0.3 to 22 μg Se/l, the biota contained whole body levels of 0.7 to 37 μg Se/g dry weight. Sequential sampling of growing indigenous fish (*Fundulus kansai* and *Pimephales promelas*) revealed no increase in whole body Se levels per unit weight as age increased. Aquaria experiments with the same fish suggested that Se levels in tissues were more influenced by Se availability in food than by Se uptake directly from water. Bican and Drbal [196] determined the content of Zn in muscle, gills, liver, spleen, kidneys, and male gonads of carp, tench, perch, pike-perch, maraena, and bream from Czechoslovakia. The kidneys accumulated the largest amount of Zn, followed by the gills and spleen. The highest values by species were found in the carp, and the lowest in pike-perch and tench. In anticipation of the development of a Pb-Zn ore

body in the vicinity of Strathcona Sound (Baffin Island, Canada), Bohn and Fallis [197] determined normal background metal concentrations in marine and freshwater fish including landlocked Arctic char (*Salvelinus alpinus*). Arsenic was detected in both muscle and liver samples, with the highest concentration (1.2 μg/g) measured in a liver sample. Cadmium was detectable only in livers and ranged up to 2.3 μg/g. Zinc concentrations in char livers were generally higher (130 μg/g) than Cu concentrations. Lead concentrations were 0.5 μg/g and below in livers. No correlations existed between body weight and metal concentration in char tissue. Atchison *et al.*[198] determined Cd, Pb, and Zn concentrations in bluegill from two Indiana lakes. Metal concentrations ranged from high levels of 15 μg Cd/g, 42 μg Pb/g, and 480 μg Zn/g to low levels of 86 μg Zn/g and nondetectable amounts for Cd and Pb. No significant effects of size on metal concentration in fish was found. The relative levels of contamination in the bluegill closely followed the relative concentrations of metals in the water and sediment at each site. Rabe and Bauer [199] measured Zn, Cu, and Cd concentrations in water, sediments, benthic insects, and fish of nine small lakes along the main stem of the Coeur d'Alene River (Idaho), which had received mining waste discharges since the 1890's. The five fish species studied could be ranked generally according to trophic level from omnivorous to carnivorous in the order: brown bullhead (*Ictalurus nebulosus*), yellow perch (*Perca flavescens*), pumpkinseed (*Lepomis gibbosus*), black crappie (*Pomoxis nigromaculatus*), and largemouth bass (*Micropterus salmoides*), with bullheads being omnivorous, largemouth bass feeding almost exclusively on fish, and the others feeding on plankton, benthic organisms, and surface insects. Zinc concentrations in muscle decreased along this rank from brown bullhead to bass, but no such trend was evident with Cu and Cd. Giesy and Wiener [200] determined the frequency distributions of whole body concentrations of Cd, Cr, Cu, Fe, and Zn among five species of freshwater fishes: chain pickerel (*Esox niger*), bluegill, blueback herring (*Alosa aetivalis*), brook silverside (*Labidesthes sicculus*), and golden shiner (*Notemigonus crysoleucas*), from a 1 120-ha impoundment near Aiken (S. C.). Species differences in mean concentrations were found for all five elements. After analyzing several types of frequency distribution models, it was concluded that in most cases either the normal or log-normal distribution may be satisfactorily used to represent frequency distribution of trace metal concentrations in fishes. In a survey of freshwater fish netted in several Polish river systems, Gajewska [201] observed a Cd content ranging from 0.01 to 0.144 μg Cd/g tissue. It was concluded that the Cd content would not exceed the allowable 400 to 500 μg Cd/person per week dose recommended by the FAO/WHO Expert Committee. Bebbington *et al.*[202] sampled nine species of Australian commercial fish for Hg, Cd, Pb, Cu, Zn, Se, and As. The fish were predominantly marine, but included several species that inhabit estuaries or rivers of varying salinity for a portion of their life cycle. Of the 232 fish analyzed for Cd, Pb, Cu, and Zn, 231 had concentrations below the Australian National Health and Medical Research Council (NHMRC) standards. Sixteen of the 232 fish exceeded the standard for Hg. Of the 95 fish analyzed for As and Se, 20 had concentrations equal to or greater than the NHMRC standard. None exceeded the standard for Se. Borg and Lithner,[203] in an investigation of increased Ni and Cd levels downstream from a Ni-Cd accumulator factory, found that Cd was selectively accumulated in fish and other biota, relative to Ni. The content of methylmercury in pike (*Esox lucius*) from the Turku archipelago on the southwest coast of Finland was studied by Linko and Terho.[204] Pike muscle contained an average of 0.27 μg Hg/g on a fresh tissue basis and exhibited the highest levels of all tissues examined, followed by liver, kidney, and gonads. Gajewska and Nabrzyski [205] determined the Hg, Cd, and Pb content of marine and freshwater fish from Poland. The average Hg, Cd, and Pb concentration for all fishes was 65, 41, and 199 ng/g, respectively. Gergely *et al.*[206] measured total content in fish from rivers and lakes from Hungary. Fish species included *Cyprinus carpio, Abramis brama, Lucioperea sandra, Amiurus nebulosus, Hypophthalamyctis molitrix et nobilis, Esox lucius, Tinca vulgaris, Perca fluviatilis, Aspius rapax, Eupomotis aureus,* and *Salmo fario*. Mercury content in fish from rivers was generally higher than that in fish from lakes and artificial ponds, and did not exceed or only slightly exceeded 0.5 μg/g, the maximum permissible level in most countries. Kempf and Sittler [207] determined Hg and organochlorine compounds in five species of fish following a mass mortality of fish in the Rhine River. The high concentration of these pollutants in fish probably explained their in-

creased mortality rate during the period 1973–1975. As part of the National Pesticide Monitoring Program of the U. S. Fish and Wildlife Service, Walsh *et al.*[208] analyzed selected fish samples from 100 monitoring stations for residues of Hg, As, Pb, Cd, and Se in 1971–1973. At most stations, detectable residues of all metals were present in more than 95% of the composite samples. The number of composite samples in which residues of these elements exceeded 0.5 μg/g decreased from 1971 to 1973, whereas the percentage of samples with detectable residues increased slightly. Koli *et al.*[209] determined the content of Cu, Fe, Zn, Mn, Cd, and Hg in brown trout, mudfish, white bass, and catfish from rivers and lakes in South Carolina. Only common names of fish species were reported. Larger fish accumulated higher levels of trace metals than small fish, and the highest levels were found in brown trout (Cu, 0.2 μg/g; Fe, 0.63 μg/g; Zn, 0.59 μg/g; Mn, 0.03 μg/g; Cd, < 0.01 μg/g; and Hg, 0.51 μg/g). Cherry and Guthrie[210] measured the concentrations of 10 toxic elements (Ti, Mn, Cu, Cr, Zn, As, Se, Co, Cd, Hg) in water, benthic sediment, plants, invertebrates, and vertebrates of an ash basin and its drainage system at a coal-fired power plant of the Savannah River Plant Project, near Aikin (S. C.). Only one fish species, the mosquitofish (*Gambusia affinis*), was found. Concentration of Zn, Cu, Cr, and Cd in the mosquitofish exceeded that found in the ash water. Historical and current heavy residues in several species of Hudson River fish were examined by Rehwoldt *et al.*,[211] using museum specimens captured in the freshwater stretch of the river system. Fish species analyzed were alewife (*Alosa pseudoharengus*), Atlantic sturgeon (*Acipensor oxyrhynehus*), fundulus killifish (*Fundulus diaphanus*), smallmouth bass (*Micropterus dolomieui*), spottail shiner (*Notropis hudsonius*), striped bass (*Morone saxatilis*), sunfish (*Lepomis gibbosus*), and white perch (*Morone americana*). The median value for the residues was independent of time. The only relationship that appeared to exist was between feeding habits and residues, with predators being higher in Hg. Lead and Cd did not seem to be dependent on feeding habits. Larson[212] compared Hg levels in fish from a eutrophic lake in Oregon to levels in fish from an adjacent oligotrophic lake. Results showed higher concentrations in fish from the eutrophic lake than from the oligotrophic lake, contrary to previously reported trends. The higher rate of assimilation was attributed to stress from pH, temperature, and DO gradients in the eutrophic lake. Fish species analyzed were cutthroat trout (*Salmo clarki*), rainbow trout (*Salmo gairdneri*), cutthroat-rainbow cross, brown bullhead, black crappie, and bluegill. Korda *et al.*[213] measured the concentrations of Cu, Zn, Mn, Na, As, Hg, Co, Sc, Cd, Se, and the rare-earth elements in the flesh and liver of two species of sculpin (*Cottus cognatus* and *Myoxocephalus quadricornis*) from areas of Lake Superior with high and low concentrations of taconite tailings. No statistically significant differences were found in the concentrations of these elements in sculpin from the two sites.

Davies *et al.*[214] determined the acute toxicity (96-hour LC50) of Ag to rainbow trout to be 6.5 μg Ag/l in soft water and 13.0 μg/l in hard water. The long-term no-effect concentration based on an 18-month exposure, but not reflecting spawning or reproduction, was estimated to be between 0.09 and 0.17 μg/l. Cadmium toxicity to the mosquitofish and to a zooplankter was compared by Giesy *et al.*[215] in soft well-water containing extremely low organic carbon concentrations of naturally occurring recalcitrant organic compounds. Even though no significant difference in the slopes of the mortality curves was found, differences in LC50 values could not be related to differences in Cd binding capacities as determined by a Cd selective ion electrode. The acute lethality of Cu over a wide range of combinations of water hardness and pH to rainbow trout was determined by Howarth and Sprague.[216] In the same study, the hypothesis that Cu toxicity was due to certain ionic species, rather than to total Cu, was tested. The 96-hour LC50 of total dissolved Cu ranged from 20 μg/l in soft acid water to 520 μg/l in hard alkaline water. Using a curve-fitting argument based on a model of Cu toxicity, ionic Cu and two ionized Cu hydroxides seemed to be the toxic species. In an evaluation of the effect of temperature on the sensitivity of food web organisms, Cairns *et al.*[217] measured the acute toxicity (24-hour LC50) of CN^-, Zn, $CrO_4^=$, Cu, and phenol between 5° and 30°C to goldfish (*Carassius auratus*), golden shiner, bluegill, channel catfish (*Ictalurus punctatus*), and rainbow trout. Survival of fish species was enhanced at low concentrations of chemicals and high temperatures, and at high chemical concentrations. Tucker and Boyd[218] examined the toxicity of $KMnO_4$ (a compound frequently used as a pond treatment to alleviate temporary O_2 depletion) to bluegills and fathead minnows

(*Pimephales promelas*). The highest concentration not toxic to fish in tap water was 2 mg/l. However, no bluegills died when treated with 5 mg $KMnO_4$/l in pond water with a $KMnO_4$ demand of 1 mg/l. Mortality of fathead minnows was negligible when treated with 12 mg $KMnO_4$/l in pond water with $KMnO_4$ demands of 3 to 5 mg/l. In order to evaluate the effect of contamination of the aquatic environment by run-off from fields after the application of Se-enriched fertilizers, Gissel Nielsen and Gissel-Nielsen [219] performed two experiments; one in which young rainbow trout were exposed to constant concentrations of Se, and one in which shorter acute exposures were followed by a period in clean water. The results showed that a chronic exposure to 100 μg Se/l or less was nonlethal but that lethality at higher concentrations depended on length of exposure. If the trout survived for the first 10 days in clean water after exposure to Se the concentration in the fish decreased by excretion to the extent that later death was not likely to be due to Se accumulation. Using static bioassays, McCarty *et al.*[220] attempted to relate mortality patterns due to Cd for the goldfish to chemical changes taking place in relatively soft and hard water throughout a 240-hour period of observation. A two-phase mortality pattern in the hard water tests indicated the likelihood of at least two modes of Cd lethality, a reduction in gas-exchange efficiency, and interactions at the enzyme and/or structural protein level. The additional possibility of a pH-related component of toxicity at higher Cd levels was discussed. Morgan [221] investigated the use of fish as a biological sensor for toxic compounds in potable water. Largemouth bass were exposed to Cu, Cd, Hg, Pb, phenol, NH_3, and CN^- while confined to continuous-flow chambers in which the frequency of opercular movements were monitored electronically. The toxicant was defined as detected when 60% or more of the fish sensors had responded. Levels of detection were, in general, between 5 and 10% of the 48-hour LC50. Lebedeva [222] examined the effect of $BeCl_2$ on survival, reproduction, and growth of aquatic organisms, including a topminnow (*Leucaspius delineatus*). At concentrations of from 0.025 to 0.8 mg Be/l, the fish survived until the end of the 150-day experimental period; however, 0.8 mg/l had a depressing effect on growth rate. A maximum water level of 0.01 mg Be/l was recommended based on tests with daphnia. The toxicity of Be and Ga to developing carp eggs (*Cyprinus carpio*) was investigated by Hildebrand and Cushman,[223] using the toxicity of Cu as a reference. Concentrations of Cu below 0.09 mg/l, Be below 0.08 mg/l, and Ga up to 2.4 mg/l did not reduce hatching success, while Cu above 0.7 mg/l, Be above 0.2 mg/l, and Ga above 17 mg/l completely inhibited hatching success. By comparing the response of a fungal parasite of carp eggs to Cr III and Cr VI compounds, with the hatchability of carp eggs at identical Cr concentrations, Draggan [224] determined that increased egg mortality and increased fungal growth were coupled at low concentrations of the compounds. A mechanism for the interaction of these chemical and biological stresses was presented. Kiokemeister and Weber [225] determined the effects of Zn and Ni chloride and their mixtures on relative growth rates of the juvenile guppy (*Poecilia reticulata*), using a two-way classification scheme that showed different types of toxicant interaction. The concentration range used to develop the dose response curve was 6.36 to 10.8 mg Ni/l for Ni, and 0.96 to 2.47 mg Zn/l for Zn. The Ni and Zn dose response curves were not statistically different from parallel, consequently concentration addition was predicted. Actual interaction in the 1:4, Zn:Ni mixture was less than predicted. Spehar *et al.*[226] investigated the toxicity of Cd and Zn mixtures to the flagfish (*Jordanella floridae*) by studying the effects of these metals on all developmental stages of the life cycle, and determined their combined effects on bioaccumulation. Survival of flagfish larvae not previously exposed as embryos was significantly reduced in all concentrations except that containing Cd alone. Survival of larvae exposed as embryos was significantly decreased in both individual Cd and Zn concentrations, but was not decreased in mixtures of these concentrations. Residues of Cd and Zn accumulated when fish were exposed to both individual and mixture concentrations and appeared to reach an equilibrium after 30 days. The uptake of one metal was not influenced by the presence of the other. Eaton *et al.*[227] determined the toxicity of Cd to embryos and larvae of seven freshwater fish species: brook trout (*Salvelinus fontinalis*), brown trout (*Salmo trutta*), lake trout (*Salvelinus namaycush*), northern pike (*Esox lucius*), white sucker (*Catostomus commersoni*), smallmouth bass, and coho salmon (*Oncorhynchus kisutch*). Species differences in sensitivity were not great, ranging over only a factor of 4 from the most to least sensitive. Larvae were consistently more sensitive than

embryos, and larval sensitivity agreed well with the results of life cycle chronic toxicity studies, indicating that embryo and larval exposures will give reliable estimates of the chronic toxicity of Cd to other fish species. The effects of Cu, Ni, dieldrin, and selected mixtures of these compounds on the relative growth and food consumption rates of juvenile guppies were analyzed by Muska.[228] Mixtures of Cu and Ni appeared to be either concentration additive or slightly supra-additive, depending on the defined response. The results of the dieldrin-Ni study were inconclusive. McKim *et al.*[229] determined the sensitivity of larvae and early juvenile stages of eight species of freshwater fish to Cu. Larvae and early juveniles of all species were more sensitive to Cu than the embryos. Embryo survival was affected only at the higher concentrations tested for all species except the rainbow trout, which was considerably more sensitive. Copper had no affect on smallmouth bass embryos at any concentration tested. Other species tested were brook trout, brown trout, lake trout, northern pike, white sucker, and lake herring (*Coregonus artedii*). The chronic effect of prespawning exposure to various concentrations of Cu on fathead minnow reproduction was investigated by Pickering *et al.*[230] The MATC was estimated to be 32 μg Cu/l, which is 0.07 of the 96-hour LC50 determined in the same study. The results showed that the length of time fathead minnows were exposed to Cu before spawning had no significant influence on egg production, but that apparently the observed reduction of egg production due to Cu levels above the MATC was the result of exposure of the sexually mature fish only during spawning. The effect of methylmercury on fin regeneration by two euryhaline fishes, mullet (*Mugil cephalus*) and killifish (*Fundulas confluentus*) was investigated by Weis and Weis.[231] Fin regeneration in the mullet was shown to be retarded by methylmercury and to be dependent on dose. Experiments with killifish indicated that while Cd or methylmercury alone can retard fin regeneration, the effect is diminished when both are present. Chan[232] investigated the chronic effects of methylmercury on the reproduction of the teleost fish (*Oryzias latipes*). Fish exposed to 43 to 1 000 μg CH_3Hg^+/l had tissue levels below 40 μg CH_3Hg^+/g, while fish exposed to more than 1 000 μg/l accumulated methylmercury to as high as 408.1 μg/g. Six-week exposure to 4.3, 10.7, and 21.5 μg/l resulted in inhibition of spawning. Japanese medaka (*Oryzias latipes*) eggs were exposed by Dial[233] to 40 to 80 μg/l methylmercuric chloride (MMC) for a period of 10 to 25 days in order to observe gross teratogenic and lethal effects during embryogenesis. The severity of defects increased with MMC concentration, leading to the conclusion that trace amounts of MMC caused various defects and presented a potential hazard to the develop...ent of embryos in aquatic vertebrates. Brenner and Cooper[234] investigated the effect of Fe $(OH)_3$ on the embryonic development of the coho salmon. Results indicated that suspended $Fe(OH)_3$, at a concentration of 3 mg/l, had no apparent effect on the hatchability, embryonic development, or survival and maturation of newly hatched coho salmon alevins. In a year-long experiment on the effects of chronic exposure of several generations of the guppy to low concentrations of NaOH and Na_2CO_3, Rustamova[235] concluded that concentrations of 25 and 50 mg/l of NaOH and Na_2CO_3, respectively, were toxic to the guppy. Speranza *et al.*[236] investigated the effect of sublethal concentrations of Zn on reproduction of the zebrafish (*Brachydanio rerio*). Adult zebrafish, when held in water containing 5 mg Zn/l for a 9-day period, during which the gametes were maturing, showed a delay in spawning. However, the effect was completely reversed by returning the exposed fish to a Zn-free environment.

Labat *et al.*[237] discussed the passive accumulation of metals by fish as related to low concentrations in the water. It was concluded that there should be three critical thresholds for metal content in water, each one affecting very different physiological processes. In a study of the accumulation of Co during embryonic development of rainbow trout, Kunze *et al.*[238] found that levels of Co concentration in the eggs were directly proportional to the Co concentration in the water. Alkaline earth metals, such as Ca, competed with Co for uptake and reduced the accumulation of Co by the fish eggs. No increase in Co was found in the larvae hatching from these eggs, indicating that accumulation took place on the egg surface, where it was probably bound by the chorion mucopolysaccharide. The study also indicated that desorption of Co takes place at a definite stage of egg development. Patrick and Loutit[239] investigated the accumulation of Cr, Cu, Mn, Fe, Pb, and Zn in a tropical fish (*Hyphessobrycon serpae*) fed on tubificid worms that had accumulated heavy metals from a diet of bacteria grown in the presence

of heavy metals. Consistent, heightened metal levels in these fish, compared to those fed an uncontaminated diet, indicated that a metal-contaminated diet would be a significant source of increased metal levels in fish. The experimental conditions were believed to model a situation below an effluent discharge point, where small concentrations of metals would be immobilized by bacteria and passed up the food chain through tubificid worms to fish. Zaba and Harris [240] examined the accumulation and effects of Ca, Mn, Zn, and Cu ions in liver mitochondria from rainbow trout, pike, cod (*Gadus morhua*), and plaice (*Pleuronectes platessa*). The mitochondria bound Ca, Cu, and Mn strongly, but Zn appeared not to be taken up to a great extent. However, Zn was the most potent inhibitor of mitochondrial respiration. By measuring the ^{45}Ca influx in perfused isolated individual hemibranches from the eel (*Anguilla rostrata*) following the removal of the corpuscles of Stannius, So and Fenwick [241] concluded that these putative endocrine glands play some role in controlling the branchial uptake of Ca. Sullivan *et al.*[242] compared the long-term, time-dependent uptake of Cd by fathead minnows under laboratory conditions to short-term uptake by the same species exposed to Cd in an industrially contaminated lake. Exposure to 2.5 μg/l in the contaminated lake resulted in accumulation of whole body burdens of 3.19 μg/g in 12 hours, while laboratory exposure to 48 μg/l in a continuous-flow laboratory system did not result in comparable levels until the eighth day of exposure. The more rapid uptake of Cd by fish in the field was thought to be due to a number of factors, including physiological stress, which increased the rate of transport of Cd into the fish. The relative importance of food and water as sources of Cd for uptake by the mosquitofish was studied by Williams and Giesy [243] in a factorially designed experiment. Water was a highly significant source of Cd throughout the 8-week exposure, but food was a significant source only after 8 weeks in the presence of Cd in water. Food was thought to become an important uptake vector only after a threshold body burden is reached, causing a decrease in the fish's ability to restrict Cd influx via gastrointestinal assimilation. Aoyama *et al.*[244] introduced a "food condition factor" into a previously developed compartmental model of the transfer of trace metals through a food chain. Simulation results from this model showed the biological dilution of metal concentration due to increase in weight of the fish. In a follow-up study, Aoyama *et al.*[245] experimentally investigated the above simulation analysis in a laboratory food chain study involving ^{137}Ce as a tracer, top minnows of (*Oryzias latipes*) as prey, and golden astro (*Astronotus ocellatus*) as predators. The results showed that the concentration of metal in predator fish increased with the ration size, that feeding interval had no effect on the uptake of the metal under the experimental conditions, and that the concentration in growing fish decreased with increased weight. These results were in agreement with the simulation. Ishak *et al.*[246] determined the distribution and tissue retention of ^{137}Cs and ^{60}Co in the Nile catfish (*Clarias lazera*) administered by ingestion of gelatin capsules and by direct uptake from water. Uptake from water, represented in terms of concentration factors, reached a maximum of 0.37 and 0.36 for Cs and Co, respectively. Internal distribution decreased in the order of muscle, bone, gill, stomach, kidneys, intestine, and liver for Cs, and in the order of bone, muscle, gills, intestine, kidneys, stomach, and liver for Co. Internal distribution following ingestion followed nearly the same order. Gothberg [247] investigated the effect of removing Hg-contaminated fish from a lake in which the Hg content of the surface sediments was low. After removal of a large proportion of the fish population, tagged pike with low Hg content were introduced in order to observe quantitative changes in muscular and liver tissues. After evaluating and comparing the ability of several mathematical models to simulate the uptake of methylmercury in bluegills, Curtis *et al.*[248] concluded that the best model was an empirical uptake relationship having the form of a rectangular hyperbola. Jernelov [249] formulated a preliminary mathematical model to account for the formation of methylmercury through microbial methylation and accumulation of methylmercury in the food chain. Accumulation in fish species directly from water and via food was included as a function of energy transfer efficiency, total biomass, growth rate and other processes. In a study of the accumulation of heavy metals from food by rainbow trout, Jacobs [250] fed rations containing Hg as methylmercuric chloride or $HgCl_2$ and Cd as $CdCl_2$. After 8 days of feeding with 7 μg Hg/g in the food, muscle tissue levels reached 1.04 μg/g, and with 1.02 μg/g in the food, muscle tissue contained 0.67 μg/g. After feeding 16 μg Cd/g in the food for over 2 months, Cd levels in muscle tissue were very small. Olson *et al.*[251] investigated

the tissue and subcellular distribution of methylmercury with respect to the lability of the methylmercury bond. Gills contained approximately 10 times as much methylmercury after 24 hours as any other tissue, whereas concentrations were lowest for brain and skeletal muscle. Following exposure, gill levels declined while levels in other tissues increased before eventually declining. In liver cytosol, a methylmercury binding metallothionein-like species accounted for up to 40% of the total bound methylmercury, indicating that methallothionein may act as a scavenger for methylmercury and inorganic Hg in fish. Sharpe *et al.*[252] examined clearance rates of methylmercury from goldfish in an attempt to quantify two of the factors controlling clearance, water temperature, and fish body size. The body content of methylmercury was cleared from the tissues of small fish at a faster rate per unit body weight than from large ones, however, no effect of temperature on clearance was observed from fish of similar body weight maintained at 5°, 10°, and 20°C. In a study of the depuration of ^{203}Hg in various trophic levels, Hamdy and Prabhu[253] determined the rate of elimination of inorganic and organic forms of Hg from the guppy. Both forms were eliminated rapidly, with a biological half-life of 6.5 days for radiolabeled mercuric nitrate and 7.2 days for labeled phenylmercuric acetate. Prabhu and Hamdy[254] measured the uptake and concentration factors (CF) of radiolabeled mercuric nitrate and phenylmercuric acetate in a simple model ecosystem consisting of bacteria, mosquito larvae, and guppies. Guppies of all ages were able to accumulate Hg from both forms. However, the CF for phenylmercuric acetate was much higher than for mercuric nitrate, which was attributed to the greater solubility of the organomercurial in lipids. Ray[255] examined the bioaccumulation of Pb in Atlantic salmon (*Salmo salar*), parr and grilse. The relative Pb level in all parr tissue samples was much higher than in the grilse. It was suggested that the relatively lower Pb level in grilse was due to either excretion in the ocean environment or to extremely rapid growth in size while the total metal load remained the same. In order to examine the bioaccumulation of PO_4^{-2} in higher trophic levels, Strange *et al.*[256] fed *Daphnia*, which had accumulated radiolabeled PO_4^{-2}, to channel catfish. Phosphorus (^{32}P) appeared to accumulate in the bone, gill, brain, and kidney, questionably in the liver, and not at all in the heart, muscle, and stomach. Catfish exposed directly to radiolabeled PO_4^{-2} incorporated the PO_4^{-2} in detectable quantities in all tissues examined. Physiological mechanisms for the observed results were discussed. The dynamics of the accumulation of ^{65}Zn chloride and nitrate by the European eel (*Anguilla anguilla*) were examined by Foulquier and Grauby.[257] Concentrations in the aquaria continuously declined, along with an uninterrupted increase in concentration in the eels. Although equilibrium with the water was never reached by the end of the experiment, distribution of ^{65}Zn in the organism was identical in all experiments, with 30% in the muscle, 15% in skin and mucus, 10% in the digestive tract, 9% in the skeleton, and 8% in the gills. Coefficients of accumulation (defined as the ratio of ^{65}Zn in fish to that in water at the end of the experiment) were lower at 7°C than at 18 and 23°C, due to decreased metabolism.

Shephard and Simkiss[258] confirmed the presence of Ca-activated ATPase enzyme in the gills of the roach (*Rutilus rutilus*). The enzyme was maximally activated by Ca^{+2} or Mg^{+2} ions but was not affected by Na^+ or K^+. *In vitro*, the enzyme was inhibited by Cu^{+2}, Pb^{+2}, Zn^{+2}, and Hg^{+2} at concentrations below 10 m*M*. In an investigation of the effects of aqueous Ca content on plasma Ca levels in goldfish, Umehara and Oguri[259] found that plasma Ca did not change in goldfish transfered from fresh water to Ca-enriched freshwater. However, plasma Ca increased markedly in goldfish transfered to one-third sea water. Bauswein[260] studied the effect of Ca on the differentiating operation of the ampulary electroreceptor of the lateral line in the brown bullhead. The normal functioning of this organ was shown to depend on a sufficiently higher concentration of Ca ions (2 m*M*/l or more) in the water surrounding the fish. However, when the effect of Ca ions was studied in animals that never had contact with water other than from northern Italy (containing 1.20 m*M* Ca/l, 1.62 m*M* Mg/l, < 2.0 m*M* Na/l, and 0.035 m*M* K/l) almost no change in the receptor properties was observed, indicating that the functional properties of these sense organs depended on the medium they are adapted to. Itazawa and Koyama[261] compared the effects of oral administration of Cd on carp and on the marine porgy (*Pagrus major*). Neither a decrease of the Ca content of serum and vertebrae nor scoliosis, all of which were noticed in carp fed on food containing 140 μg Cd/g or more for 30 days, were observed in the porgy. The

fact that the porgy did not suffer from low Ca and vertebral abnormalities, even though fed 7 to 11 times as much Cd as the carp, was considered to be at least partly attributable to Ca derived from the sea water. The relationship between Cd accumulation in fish and the mechanisms of the Na-K pump were investigated by White.[262] Rainbow trout were exposed to concentrations of 20, 10, and 0 μg Cd/l for 30 days. Preliminary results indicated that bioaccumulation of Cd in the liver and kidney was related to concentration and exposure time. No significant changes were observed in liver and kidney ATPase activity, Na^+ or K^+ levels, or water content. Christensen *et al.*[263] measured the biochemical response of brook trout to experimental water concentrations of methylmercuric chloride, $CdCl_2$, and $PbNO_3$. Significant increases in plasma Na and Cl^-, and decreases in hemoglobin and glutamic oxalacetic transaminase activity were caused by Pb; increases in plasma Cl^- and lactic dehydrogenase activity, and a decrease in plasma glucose was caused by Cd. Increases in hemoglobin and plasma Na and Cl^- were caused by methylmercury. The potential interference of Cd with the normal hepatic storage of vitamin B_{12} in the immature pumpkinseed (*Lepomis gibbosus*) was investigated by Merlini.[264] Experimental results showed that untreated control fish were able to store dietary vitamin B_{12} in the liver, whereas fish in water containing 40 μg Cd/l had statistically less vitamin in their livers and more in the gall bladder, intestinal tract, and gills. Schreck and Lorz[265] examined the potential use of serum cortisol elevation as an indicator of stress by exposing coho salmon to Cu and Cd. The two meals appeared to have completely different modes of action in eliciting a general stress response in coho salmon. Exposure to Cd caused death of yearling salmon without elevating their cortisol levels, while Cu exposure was followed by a large, dose-dependent cortisol response. In order to clarify whether the interference of Zn metabolism by Cd could be correlated with modification of the activity of Zn metallo-enzymes, Piavaux[266] exposed pumpkinseed sunfish to different concentration of Cd. The activity of alkaline phosphatase was determined in various organs of control and Cd-treated fish, along with Cd and Zn concentrations. The activity of the enzyme did not follow any systematic trend; therefore, there was no apparent competition for active metabolic sites of alkaline phosphatase in favor of Cd. Roales and Perlmutter[267] observed an apparent decrease in the immune response of the blue gourami (*Trichogaster trichopterus*) to both infectious pancreatic necrosis virus and to *Proteus vulgaris*, on exposure to 9 μg/l of methylmercury, Cu, or methylmercury plus Cu. Fish exposed to both toxicants did not appear to have any greater or lesser immunological response to either antigen than fish exposed to either toxicant individually. In a series of three papers, Yamamoto *et al.*[268-270] reported the results of studies on Cu metabolism in fishes. The results indicated that L-ascorbic acid (AsA) prevented the accumulation of Cu and that accumulated Cu decreased the AsA level in the hepatopancreas by inhibiting AsA biosynthesis. A statistically significant increase was observed in the hepatopancreas, gills, intestine, and kidney after carp were exposed to 0.1 mg Cu/l for 2 weeks. The highest Cu content was in the hepatopancreas and was determined to be bound to the relatively lower molecular weight protein fraction. This Cu-binding protein was determined to be a metallothionein-like protein, which is apparently synthesized in the carp hepatopancreas on induction by Cu. The effect of Cu exposure on the immune response of juvenile coho salmon was examined by Stevens.[271] Significant mortality occurred among coho fingerlings exposed to 24.6 μg Cu/l and higher for 31 days, and most of the survivors were unable to adapt to sea water. The antibody level against *Vibrio anguillarum* was significantly reduced in fish exposed to 18.2 μg Cu/l compared to control animals. The efficacy of $CuSO_4$, $KMnO_4$, and Diuron in controlling external bacterial infections of bluegills was investigated by Phelps *et al.*[272] Potassium permanganate at 3 mg/l proved to be effective in significantly reducing fish mortality. Both the higher (5 mg/l) and lower (1 mg/l) concentrations of $KMnO_4$ and the other treatment chemicals were ineffective in significantly reducing infection. Sastry and Gupta[273] exposed the fresh water fish *Channa punctatus* to a sublethal concentration (3.80 mg/l) of $PbNO_3$ in order to observe whether chronic treatment produced any alterations in the activity of the digestive enzymes. A decrease in the activity of alkaline phosphatase was recorded after 15 days, but no significant change after 30 days. Acid phosphatase was elevated after both periods. Amylase, maltase, and lactase showed elevation after 15 days, followed by an inhibition after 30 days. Pepsin and trypsin activities remained above normal throughout the duration of the exposure. In

a study of the chronic toxicity of Pb to rainbow trout, Hodson *et al.*[274] demonstrated that waterborne Pb was readily taken up by fish resulting in subtle, sublethal, and physiological responses. In contrast, dietary Pb was not taken up at all and did not affect fish. Exposure to lead in water at concentrations as low as 13μg/l caused significant increases in red blood cell (RBC) numbers, decreases in RBC volumes, decreases in RBC cellular iron content, and decreases in RBC delta-amino levulinic acid dehydratase activity. The effect of test water pH on the concentration of Pb in the blood of juvenile rainbow trout was also studied.[275] A decrease in pH of 1.0 unit from any reference point in the range of 10.0 to 6.0 resulted in an increase in blood Pb by a factor of 2:1. Results indicated that blood Pb was in equilibrium with water Pb. Possible mechanisms for the observed relationship, including a change in the availability of Pb complexes or increased permeability of fish tissue, were discussed. Panigrahi and Misra[276] examined some toxicological effects of mercuric nitrate on the freshwater fish *Anabas scandens*. All fish exposed to 3 mg/l mercuric nitrate survived, but showed a variety of pathological and biochemical disorders, including blindness. A total of 71% had become blind after 4 weeks of exposure. Physiological and biochemical disorders were related to the concentrations of Hg in the tissue, which reached 3.0 μg/l wet tissue in the liver and 2.8 μg/g wet tissue in the muscle. Fromm[277] reported on a number of studies designed to explore the mode of action of pollutants on fish. Studies of the effect of inorganic and organic Hg on rainbow trout indicated that uptake was primarily through the gills in nonfeeding fish. Use of perfused gills appeared to be a sensitive model to detect deleterious action of pollutants on fish. Strange *et al.*[278] exposed channel catfish to elevated levels of PO_4^- and monitored cardiac and opercular rates by means of implanted electrodes. Results suggested that cardiac and opercular rates increased with increasing PO_4^- concentration up to approximately 12 mg/l. Concentrations up to 160 mg/l failed to cause further increases in cardiac rhythm. However, exposure to 20 mg/l for 2 weeks caused a gradual increase in cardiac rate. Goudie and Davis[279] compared tissue Na and Cl^- levels and intra-extracellular water distribution in channel catfish acclimated to NaCl solutions with and without Ca enrichment. In NaCl solutions up to 11 g/l without Ca, slight elevations in plasma Na and Cl^-, Cl^- space, and muscle Na occurred, but muscle water decreased compared to those with no added Na. Fish held in Ca-enriched tap water revealed no apparent differences in these parameters at 0 and 7.5 g NaCl/l, and less of an elevation at 11 g NaCl/l than without Ca. Criswell *et al.*[280] examined the effect of environmental Ca on the regulation of plasma Na and Cl^- in channel catfish. Plasma chloride levels were significantly lower in fish from deionized (98.0 meq/l) water and aged tap water (96.3 meq/l) than in fish held in Ca-enriched tap water (117.8 meq/l). Plasma Na of fish from aged tap water (117.1 meq/l) was significantly lower than in fish from deionized (135.8 meq/l) and Ca-enriched (131.3 meq/l) tap water. Mortality of catfish exposed to a Ca-free solution of 12 g/l NaCl was 87.5% after 48 hours, but mortality in groups with Ca added to the salt water was negligible. To clarify the mode of action of Zn toxicity, Hughes and Adeney[281] compared the effect of environmental hypoxia on the respiratory and cardiovascular physiology of rainbow trout before and after an extended treatment with 40 and 10 mg Zn/l. The changes that occurred were similar to those which are known to occur when trout are subjected to environmental hypoxia, leading to the conclusion that Zn interferes with some of the mechanisms involved in the uptake of O_2 by the gills. Ogino and Yang[282] investigated the requirement of rainbow trout for dietary Zn by feeding them diets containing graded levels of Zn. The results demonstrated that Zn is an essential trace element in the diet of rainbow trout, even though the water contained 11 μg/l. The feeding of rainbow trout with a diet deficient in Zn resulted in an extremely low growth rate, high mortality, and the appearance of deficiency symptoms such as cataracts in the eyes and erosion of the fins and skin. The growth rate was restored to normal by the addition of $ZnSO_4$ to the deficient diet. Adragna and Privitera[283] examined cell population densities, protein content, cellular diameters and volumes, and Zn content and accumulation in fathead minnow cell cultures in response to variations in Zn concentration in the culture medium. They found that concentrations of Zn, which were lethal to naive cells, were not lethal to cells which were slowly adapted to higher levels of Zn, suggesting an activation of increased synthesis of a protein-binding factor for Zn, an increased metalloenzyme production, and an increased synthesis of structural proteins. Cells in which these modifica-

tions had taken place could more effectively handle the Zn stress.

Waiwood and Beamish [284] determined the effects of Cu, pH, and hardness on the critical swimming speed of rainbow trout, defined as the maximum velocity fish could maintain for a precise time period. In the absence of Cu, critical speed was not appreciably influenced by hardness, pH, or time of exposure. For any given hardness, Cu had a greater effect at low than at high pH. In order to determine if $CuSO_4$ might stimulate feednig activity of fishes, which might be useful in expediting the intake of a drug mixed with food, Morrice [265] exposed roach to $CuSO_4$ at 0.5 and 0.7 mg/l. These concentrations were shown to decrease food consumption, and hence were not useful for this purpose. In a model ecosystem designed to mimic a natural environment, Sullivan *et al.*[286] and Kwasnik [287] demonstrated that fathead minnows showed increased vulnerability to predation by largemouth bass after chronic exposure to Cd at 0.025 mg Cd/l, which is well below the reported MATC for fathead minnows. The lowest acute concentration producing increased prey vulnerability was 0.375 mg/l. The increased prey susceptability was attributed to altered behavioral patterns, including abnormal schooling behavior. Using a quantitative index of the swimming activity of the bluegill, Ellgaard *et al.*[288] investigated the effect of Cd, Cr, and Zn on locomotor activity. Hyperactive locomotor responses were found to be affected in a concentration-dependent relationship. Cadmium altered locomotor activity more than Cr or Zn. Hartman [289] investigated the effects of Hg feeding schedules on accumulation and retention of Hg in rainbow trout and on trout behavior. Trout were administered Hg in the food in the form of Ceresan (ethyl mercury p-toluene sulfonalide). Both dose level and schedules influenced the concentrations of Hg in muscle. Fish receiving 10 μg/g or more of Hg every 5 days or 5 μg/g or more every day were unable, with few exceptions, to learn to avoid shock when preceded by a signal light. There was no evidence of any other behavioral impairment nor any indication of physical debilitation resulting from any treatment. Bloom *et al.*[290] found that female zebrafish exposed to 5 mg Zn/l do not respond to female aggregating pheremone at a concentration which attracted nonZn-exposed females. A mechanism for this effect was hypothesized which involved an effect on the nasal or gustatory epithelium. The effects of Zn and Cu on the survival of the common carp and the grass carp (*Ctenopharyngodon idellus*) were investigated by Wong *et al.*[291] The common carp was found to be more susceptible to Cu, whereas the grass carp was more susceptible to Zn. Histopathological examination of the gill and liver of the common carp revealed particles around the gills of dead fish treated with Cu and Zn salts. The internal tissue and the liver also appeared to be damaged.

Organics. Armstrong and Lutz [292, 293] analyzed samples of fish obtained from Lake Superior and Lake Huron in 1974 for PCBs, chlorinated insecticides, other organic compounds, heavy metals, and radioactivity. Lake trout (*Salvelinus namaycush*) and siscowet (*Salvelinus namaycush siscowet*) from Lake Superior were higher in PCB, DDT, and Hg than other fishes, while slimy sculpin (*Cottus cognatus*) and lake herring (*Coregonus artedii*) were lowest. PCB content was above the Canadian limit of 2 μg/g in lake trout from three locations of Lake Superior; DDT was above Canadian and United States limits of 5 μg/g in lake trout from three locations, and Hg was at or above the Canadian limit of 0.5 μg/g in lake trout from six locations and in burbot (*Lota lota*) from one location. PCB content above the present Canadian limit of 2 μg/g was found in one pooled sample of bloater chub (*Coregonus hoyi*) and in one of burbot from Lake Huron. All samples from Lake Huron complied with Canadian and United States regulations for DDT, and Hg and radioactivity in these samples was low. Glass *et al.*[294, 295] monitored the loadings of various organic compounds present in water, sediment, and fish samples collected from Lake Superior and Lake Huron. The various contaminants were evaluated on the basis of degradability, persistence in the aquatic environment, and the potential to cause taste and odor problems in water and fish flesh. Problems or potential problems associated with the levels of each organic contaminant found to be present in Lake Superior and Lake Huron water, fish, or sediment samples were discussed. Veith *et al.*[296] measured the residues of PCBs and DDT in 10 species of fish, zooplankton, and sediment from western Lake Superior. Fish sampled in 1972 to 1973 contained total DDT and PCB residue concentrations greater than 0.1 mg/l, and many of the larger fish contained residues that exceeded the 5 mg/l tolerance level established by the U. S. Food and Drug Administration. The apparent bioconcentration factor for PCBs in

Lake Superior fish ranged from 10^5 to 10^6. Spagnoli and Skinner [297] assessed the level and extent of PCB contamination in 52 species of fish and six species of shellfish inhabiting or migrating through major waterways in New York State. Nearly all fish collected during 1970 to 1975 contained PCBs in detectable amounts, although the levels of contamination and the specific Aroclor varied. Fish from the Hudson River contained the highest known PCB concentrations within the United States, with some levels exceeding 100 mg/l. Other waters and fish that were significantly contaminated with PCBs include Lake Ontario salmonids, Cayuga Lake lake trout, Onondaga Lake channel catfish, and American eels. The PCB levels in roach (*Leuciscus rutilus*) from Lake Roxen (Sweden) were monitored up to 12 times a year for 7 years by Olsson *et al.*[298] to determine if seasonal variation in PCB levels occurred. In most instances, PCB levels in roach were found to peak suddenly in the spring and then decrease rapidly back to a base level. Several explanations for this phenomenon were discussed. Hattula *et al.*[299] conducted a 5-year monitoring study of chlorinated hydrocarbon concentrations present in 12 species of fish sampled from five different areas of Lake Päijänne (Finland). Considered without reference to species, areas, or research years, the average PCB concentration was 0.056 μg/g, and the average DDE and DDT concentration was 0.010 and 0.006 μg/g, respectively. The average values of lindane, aldrin, dieldrin, and DDD were under 0.0005 μg/g. Concentrations of PCBs in the tissues of fish, sediment, and water from Lake Hartwell, Lake Weiss, and the Coosa River were reported and analyzed by Bruner and Hill.[300] STORET data and PCB levels were analyzed for eight states to define potential problem areas. The PCB problem in the southeastern United States was evaluated and recommendations regarding future sampling needs were made. Moccia *et al.*[301] discussed possible explanations for the increasing frequency of thyroid goiters observed in coho salmon (*Oncorhynchus kisutch*) from Lakes Michigan, Ontario, and Erie. It appeared that other factors, in addition to iodine deficiency, were involved in the etiology of the goiters. Organochlorine compounds such PCBs, DDT, dieldrin, and mirex were suggested as possible causes. Thomann [302] developed a model for describing the transport and fate of hazardous substances in the freshwater environment which took into account the size of the organism. Laboratory and field data from the literature were used for preliminary estimates of the model coefficients. Using this model, the analysis of some previously published PCB data in Lake Ontario indicated that about 33% of the observed 6.5 μg/g of PCBs in coho salmon was due to transfer from lower levels in the food chain and about 67% from direct water uptake. The model showed rapid accumulation of PCB with organism size mainly due to decreased excretion rates and decreased biomass at higher trophic levels. Westöö and Norén [303] identified the pollutants, 2',3,4,4',5-pentachloro-2-aminodiphenyl ether and 2',3,-4,4',5,6-hexachloro-2-aminodiphenyl ether, in pike (*Esox lucius*)and eel (*Anguilla anguilla*) from the Viskan River (Sweden). The highest level found (sum of both pollutants) was 0.4 μg/g in eel. The structure toxicity relations of polychlorobornane components of toxaphene were examined by Saleh *et al.*,[304] using previously published data on mice, houseflies, and goldfish (*Carassius auratus*). A major portion of the acute toxicity of toxaphene and for up to 23% of the analyzed composition of toxaphene was accounted for by 2,2,5-Endo, 6-exo, 8,9,10-heptachlorobornane, and four derivatives of this heptachlorobornane, with an additional chlorine atom at position 3-exo-, 8,9, or 10.

Verma *et al.*[305] conducted static bioassays, with five detergent formulations, using the test fish *Macrones vittatus*. The detergent containing aromatic alkyl benzene sulfonate was the most toxic (96-hour LC50, 0.29 mg/l) and the least toxic (96-hour LC50, 1.53 mg/l) contained odorless SLS. The toxicities of the remaining three detergents fell between these extremes. Rieger [306] stated that the toxicity of Austrian detergents to carp fry was directly related to the percent of active components. The greatest effect was shown by dodecyl benzolsulphonate (ABS) which caused 40% mortality at a concentration of 7.5 mg/l. The acute toxicity of linear alkylbenzene sulfonate (LAS) to rainbow trout (*Salmo gairdneri*) was evaluated both before and after degradation in an activated sludge process by Alabaster.[307] LAS (untreated) added to detergent-free (control) effluent and diluted with hard water had a nominal 4-day LC50 of 0.36 mg/l; whereas, after treatment, the corresponding value was 29.5 mg/l expressed in terms of the concentration of surfactant in the influent to the activated sludge treatment units. Wienberg [308] exposed embryos, larvae, and fry of the cyprinodontid (*Rivulus cylindraceus*) to

various sublethal concentrations of the branched alkyl benzene sulfonate (TBS) and investigated effects on mortality rates, deformities, hatching, and activity. Under optimal environmental conditions, concentrations of TBS up to 24 mg/l appeared to have no effects on larval or young fish stocks. Acute lethality and sublethal effects of acetone, ethanol, and propylene glycol on the cardiovascular and respiratory systems of rainbow trout were examined by Majewski *et al.*[309] The 24-hour LC50 values for acetone and ethanol in flow-through tests were 6 100 mg/l and 11 200 mg/l, respectively. Static exposure to 50 000 mg/l propylene glycol produced no mortality to trout in 24 hours. Acetone exposure increased the ventilation rate and buccal pressure amplitude in trout, whereas ethanol caused a slight depression of these parameters during initial stages of the 24-hour exposure. Propylene glycol exposure had a mildly stimulatory effect on ventilation rate and heart rate in adult trout. Dave and Lidman [310] conducted range-finding acute toxicity tests on 12 solvent extraction chemicals including alphatic amines, acids, alcohols, and organophosphorus compounds with rainbow trout. The 96-hour LC-50s for trout ranged from 0.18 to 56.0 mg/l based on the amounts added. Other toxic effects and behavioral changes in the fish caused by exposure to these chemicals were discussed. Acute tests with the golden orfe (*Leuciscus idus melanotus*) were conducted using 200 selected chemical compounds under comparable conditions in two different laboratories in Germany by Juhnke and Lüdemann.[311] The LC50 values for mostly organic chemical compounds ranged from 0.02 to 24 mg/l for chlorbenzol, at each respective laboratory, to 8 930 to 12 220 mg/l for diacetonalcohol. The acute toxicity of perchloroethylene, trichloroethylene, 1,1,1-trichloroethane, and methylene chloride to fathead minnows (*Pimephales promelas*) was determined, using both static and flow-through exposures, by Alexander *et al.*[312] The flow-through toxicity values of all compounds were less than the static toxicity values. Perchloroethylene was the most toxic of the chlorinated solvents tested and methylene chloride was the least toxic (Table I). In addition, concentrations of these compounds' effects on equilibrium, melanization, narcosis, or normal gill appearance in 50% of the fish 50% effective concentration (EC50s) were calculated for various time periods during the flow-through tests. The acute toxicities of hydrazine, unsymmetrical dimethylhydrazine (UDMH), Aerozine-50, and monomethylhydrazine (MMH) to guppies (*Poecilia reticulata*) were determined by Slonim.[313] Static bioassays in both hard and soft water showed that hydrazine was the most toxic of these hydrazine compounds tested and UDMH was the least toxic to guppies. Hydrazine was 6.3 times more toxic in soft than hard water; whereas UDMH was 2.6 times more toxic in hard water than soft water. When results of tests with pre-exposed guppies were compared with those of previously unexposed fish, it appeared that the toxic effects of hydrazine compounds were cumulative. The effect of methanol on the fertilization of chum salmon (*Oncorhynchus keta*) ova was investigated by Craig *et al.*[314] During both a 32-second exposure of the gametes and a 32-minute exposure of fertilized eggs, methanol concentrations up to and including 1% (by volume) did not significantly affect fertilization success, survival to hatching, hatching time, alevin size at hatching, or physical deformities among alevins. A 10% methanol concentration caused significant mortality at both exposure periods. Grieco *et al.*[315] investigated the carcinogenicity and acute toxicity of dimethylnitrosamine (DMN) in rainbow trout. Mean body weights of trout in all DMN-fed groups were significantly lower than those of the controls at all sampling dates. A direct dose-related response of hepatocellular carcinoma occurred in trout fed 200, 400, and 800 mg DMN/l at 52 weeks, and a greater incidence of carcinomas was observed at 78 weeks, even though DMN-feeding was discontinued at 52 weeks. The acute 10-day LD50 after intraperitoneal injection of DMN was 1 770 μg/g. The toxicity and teratogenicity of aflatoxin B_1 (AFB_1) in Japanese medaka (*Oryzias latipes*) eggs was described by Llewellyn *et al.*[316] Lethal effects were observed in all concentrations of AFB_1 above 0.05 μg/ml. AFB_1 exposure also resulted in teratogenic effects on the circulatory system, on several internal organs, and on normal appendage development in the medaka. The dominant lethal effects of triethylenemelamine (TEM) in the guppy were studied by Mathews *et al.*[317] Both the injection and the emersion tests resulted in higher percentages of dead embryos and lower total number of embryos with increasing doses of TEM. However, the injection method of treatment had a more deleterious effect on both percentage of dead embryos and total number of embryos than the emersion method.

Dequidt *et al.*[318] examined the short-term

effects of feeding Pyralene 3010 to young trout. No valid conclusions of Pyralene toxicity were obtained because of problems with maintaining the fish. Because of the different concentrations of Pyralene found in dead fish in the same interval of time, a defect in assimilation and a denutrition having as its origin the "plastification" of the food which impeded the action of the digestive enzymes was suspected. Hodgins *et al.*[319] tested the effects of chronic dietary exposure to crude oil on rainbow trout reproduction. Mean egg survival through hatching and survival of fry from hatching to the swim-up stage were not significantly effected in offspring of petroleum-fed fish. In addition, no morphological or histological abnormalities were observed in offspring of crude oil-fed trout. It was concluded that no significant impairment of reproductive success occurred from this type of dietary exposure to petroleum. The effects of feeding dry food soaked with silicone fluid to young rainbow trout were assessed by Mann *et al.*[320] Ingestion of siliconic food had no toxic effects on trout at the levels tested. Burks and Wilhm [321] conducted on-site continuous-flow fathead minnow and benthic macroinvertebrate bioassays to measure the toxic effects of three petroleum refinery effluents and to determine the effectiveness of several methods of treatment on the toxicity of these effluents. Bioassay results indicated that sequential biological treatment dual media filtration-activated carbon adsorption, the equivalent of EPA's Best Available Technology Economically Achievable (BATEA), produced an effluent that caused no acute lethal effects on fathead minnows or benthic macro-invertebrates. In all but one evaluation there was less than 15% mortality of the fathead minnows continuously exposed to the sequential BATEA treated oil refinery wastewater for 32 days. Barnett and Toews [322] determined the effects of crude oil and the dispersant (Oilsperse 43) on respiration, coughing rates, and acute toxicity in Atlantic salmon (*Salmo salar*). The 96-hour LC50 of a 1:1 oil:dispersant emulsion was 99 mg/l. The coughing rates of fish was significantly increased at all the sublethal concentrations tested, while respiration rates declined at the higher sublethal levels tested. Nevins and Johnson [323] determined the acute toxicity of three phosphate ester hydraulic fluids (Houghtosafe 1120, Pydraul 50E, and Pydraul 115E) to several species of freshwater fish and invertebrates. During both static and flow-through tests on rainbow trout, fathead minnows, channel catfish (*Ictalurus punctatus*), and bluegills (*Lepomis macrochirus*), Pydraul 50E was more toxic than Houghtosafe 1120. Pydraul 115E was the least toxic of the three compounds. Rainbow trout were the most sensitive fish species to all three phosphate ester mixtures. Toxicity tests with Pydraul 50E and three of its components were conducted by Sitthichaikasem.[324] Static 96-hour LC50 values for rainbow trout sac-fry, fingerlings, and bluegills exposed to these compounds are listed in Table 1. In chronic flow-through tests, Pydraul 50E reduced growth, backbone collagen concentrations, and calcium and phosphorus levels in rainbow trout, but increased whole body vitamin C concentrations. The uptake of ^{14}C-labelled tri-p-cresyl phosphate and ^{14}C-labelled tris (2,3-dibromopropyl) phosphate by bluegills was rapid, with plateau levels approached by 7 days and with accumulation factors of 1 589 and 684, respectively. Jenkins *et al.*[325] determined the acute and chronic toxicity and the uptake and elimination of the synthetic jet fuel (JP-9) and its components (RJ-4, RJ-5, and MCH) for several species of fish. The acute toxicity of emulsions, unemulsified fuel, and fuel components was determined for golden shiners (*Notemigonus chrysleucas*) (Table 1). Continuous-flow bioassays on rainbow trout yielded 97-day LC50 values for RJ-4 and RJ-5 of 0.045 mg/l and 0.072 mg/l, respectively, and 23-d LC50 values for JP-9 and MCH of 0.26 mg/l and 1.3 mg/l, respectively. Both RJ-4 and RJ-5 were concentrated in rainbow trout flesh to a much higher degree than was MCH in flagfish (*Jordanella floridae*) flesh. Voiding of MCH from fish bodies occurred readily in fuel-free water, but RJ-4 and RJ-5 appeared to be retained in the tissues of these fish. Mauck *et al.*[326] examined the effects of PCB (Aroclor 1254) on growth, survival, and bone development in brook trout (*Salvelinus fontinalis*). Growth of fry was significantly decreased after 48 days of exposure to concentrations of 1.5 μg/l and above, but no significant differences in growth of surviving fry were observed at the end of the 118-day exposure. Significant mortality occurred at concentrations of 3.1, 6.2, and 13.0 μg/l after 118 days of exposure. Exposure to Aroclor 1254 decreased hydroxyproline and vitamin C concentrations in sac-fry and decreased collagen and phosphorus concentrations in the backbones of these fish. After 118 days of exposure, Aroclor 1254 had accumulated in the surviving fry at levels from 40 000 to 48 000 times the water concentration. The acute and

chronic effects of PCB (Aroclor 1248 and 1260) on fathead minnows and the bioaccumulation of PCB residues in these test fish were determined by DeFoe *et al.*[327] The LC50 values and bioconcentration factors (BCF) for these exposures are shown in Table 1. Reproduction in fathead minnows occurred at all tested concentrations of Aroclor 1248 and 1260 up to 3.0 and 2.1 μg/l, respectively. Exposed fish placed in clean water for 60 days eliminated less than 18% of the accumulated body burden of Aroclor 1248 and 1260. Yockim *et al.*[328] measured the distribution and toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and 2,4,5-trichlorophenoxy acetic acid (2,4,5-T) in an aquatic model ecosystem, which contained soil treated with either TCDD or 2,4,5-T, water fleas, snails, algae and fish. Bioaccumulation ratios as high as 26 and 4 875 were obtained for mosquitofish (*Gambusia affinis*) in the 2,4,5-T and TCDD ecosystems, respectively. No toxic effects were observed on any organisms in the 2,4,5-T ecosystem, while 100% mortality of both mosquitofish and channel catfish occurred in the TCDD ecosystem. The chronic oral toxicity of TCDD to rainbow trout was reported by Hawkes and Norris.[329] Consumption of food containing 2.3 μg/l TCDD caused no mortality, no reduction in food consumption or growth, and no fin erosion. Consumption of diets containing 2.3 mg/l TCDD caused an average mortality of 50% in 61 days, reduced feeding activity and growth, and increased fin erosion and liver pathology. Examination of several fish, fed diets containing 2.3 μg/l or 2.3 mg/l TCDD, revealed whole body residues that indicated TCDD was not concentrating in body tissues.

The uptake, metabolism, and elimination of ^{14}C-naphthalene and ^{14}C-2-methylnaphthalene by rainbow trout were studied by Melancon and Lech.[330] Napthalene was rapidly taken up by trout and the ^{14}C levels in the tissues studied were from 22 to 340 times the initial water level of napthalene after 8 hours of exposure. The half-lives of elimination of naphthalene from all tissues except fat were less than 24 hours. The BCFs for 4-week exposures to both compounds are shown in Table 1. For both the hydrocarbons, bile contained 65 to 70%, liver 5 to 10%, and muscle less that 1 percent metabolites. The 2-Methylnaphthalene was taken up more rapidly by trout (initially accumulated to higher levels in all tissues studied) stored in muscle to a greater extent as parent compound, and eliminated more rapidly from muscle, liver, and blood than was naphthalene. Kühnhold and Busch[331] compared the difference in accumulation of carbon radioactive naphthalene, 3,4-benzopyrene and n-hexadecane in whole salmon eggs (*Salmo salar*) and in single dissected parts of these eggs. The uptake process for these three hydrocarbons was very rapid and was quickest for naphthalene and slowest for hexadecane. Measurements of accumulated hydrocarbons in the dissected parts of the embryo showed that the major amounts of the totally accumulated hydrocarbons were concentrated in the yolk and the periviteline fluid, with the exception of hexadecane, where the amount retained in or on the chorion after 3 days was approximately 50%. The accumulation and metabolism of ^{14}C labelled benzene, naphthalene, and anthracene by young coho salmon was determined by Roubal *et al.*[332] Regardless of whether these hydrocarbons were administered in the food or by intraperitoneal injection, the accumulated ^{14}C (percent administered dose) in key organs increased in the order: anthracene > naphthalene > benzene over various time periods. Determination of the metabolic fate of these hydrocarbons in the tissues of coho salmon revealed that the highest percentages of metabolites occurred in the gallbladder, while significant amounts also occurred in the liver, brain, flesh, and carcass. Sanborn *et al.*[333] examined the distribution, elimination, and degradation of [^{14}C] HCB in green sunfish (*Lepomis cyanellus*) after feeding HCB-contaminated food. The highest residues were in the alimentary tract of the fish, the lowest residues in the skeletal muscle. From half-life determinations of HCB, pentachlorophenol, and total ^{14}C in tissues sampled at days 14 and 28 after feeding, it appeared that several weeks would be required to eliminate residues accumulated during the 3-day exposure. The effects of various concentrations of Tween 20 on the amount and rate of accumulation of tris (2,4-di-ter-butylphenyl) phospate in killifish (*Oryzias latipes*) were investigated by Sugiura *et al.*[334] An apparent equilibrium of tris-phosphite uptake was reached by the fourth day for Tween 20 at 1, 5, and 10 mg/l, but was not reached by the twentieth day for Tween 20 at 100 mg/l. The accumulation amount and rate varied with the concentration of Tween 20 if it was added at concentrations of tris phosphite above its solubility. Matthiessen[335] studied the uptake, metabolism, and excretion of radiolabelled N-tritylmorpholine (Frescon) in the tropical food fish *Sarotherodon mossambicus*. Scintillation counting of tissues from adult fish

exposed to 0.005 mg Frescon/l for 24 hours showed that approximately 40% of the absorbed activity was found in bone, 16% in muscle, 16% in gut, and 10% in gill. Fish had excreted 50% of the absorbed ^{3}H Frescon after 55 hours in flowing Frescon-free water. Some effects of exposing rainbow trout to sublethal phenol concentrations were investigated by Swift.[336] Nonlethal phenol concentrations had no effect on the urine flow rate or hematocrit of trout after exposure for 24 hours. Both the uptake of phenol into tissues and the loss of phenol after exposure and subsequent transfer to clean water were rapid. The equilibrium concentration for muscle was reached in 3 hours and was similar to the phenol concentration to which the fish were exposed. Concentrations of phenol present in muscle, blood, and brain tissues after 24 hours of exposure to various water concentrations of phenol were given. The concentrations of PCP present in the water, suspended particulates, sediments, leaf litter, and unspecified fish species of a stream and nearby lake accidently polluted with PCP were monitored by Pierce *et al.*[337] PCP levels in the water column and suspended particulate matter were low at all sampling periods, whereas concentrations of PCP persisted in sediment samples and leaf litter for at least 17 months following contamination. Fish contained high concentrations of PCP for up to 6 months after the spill, but only trace amounts of PCP after 10 months. The accumulation of PCBs (Clophen C) in the organs of rainbow trout fingerlings was studied by Braun and Meyhoefer.[338] Residue levels were 0.62 and 12.3 μg/g in gills, 0.82 and 7.6 μg/g in muscle, and 3.47 and 10.6 μg/g in liver (wet weight) after 8 weeks of exposure to 2 and 20 μg PCB/l, respectively. Residues in tissues from trout held in PCB-free water for 10 weeks had decreased to 1/2 in liver and to 0 in gills, but showed no degradation in muscle. The accumulation of 10 PCB and PBB compounds in the killifish was investigated by Sugiura *et al.*[339] to determine if the resulting accumulation factors were proportional to the partition coefficients of these compounds. Equilibrium of accumulation was not reached during 20 days of testing. A proportional correlation was present between accumulation factors and partition coefficients when partition coefficients were below but not above 10^{6}. The accumulation rate decreased with increasing partition coefficients in the compounds with partition coefficients up to 10^{6} and apparently increased above 10^{6}. Zitko and Carson[340] studied the uptake and excretion of 2,4,4′-tri-chlorodiphenyl ether, 2,3′,4,4′-tetrachlorodiphenyl ether, 2,2′,4,4′,5-pentachlorodiphenyl ether, tetrabromo-2-chlorotoluene, and pentabromotoluene in juvenile Atlantic salmon exposed to mixtures of these compounds in water and in food. Accumulation coefficients and half-life values for the individual chlorinated diphenyl ethers and brominated toluenes were calculated and compared with those calculated for 2,4′,5-tribromobiphenyl and 2,2′,4,5′-tetrabromobiphenyl, which were run as reference compounds. Brominated toluenes accumulated about 10 times less than chlorinated diphenyl ethers but were excreted more slowly. The hepatic accumulation, metabolism, and biliary excretion of the organic anion sulfobromophthalein (BSP) by rainbow trout was studied by Gingerich *et al.*[341] Hepatic accumulation of BSP was rapid, with more than half of the injected dose being present in the livers of trout after only 15 minutes. As many as six separable fractions of BSP and BSP metabolites were found in the bile of trout by thin-layer chromatography. Biliary excretion appeared to be the rate-limiting step in the overall transfer of BSP from plasma to bile. The effect of carbon tetrachloride (CCl_4) on hepatic accumulation, metabolism, and biliary excretion of BSP in rainbow trout was also studied by Gingerich *et al.*[342] Hepatic BSP accumulation was reduced in trout treated with CCl_4 despite higher plasma concentrations of BSP. Bible flow rates, bile BSP concentrations, the rates of biliary BSP excretion, and the separable metabolite fractions of BSP present in plasma, liver, and bile were not significantly affected by CCl_4 treatment. Statham *et al.*[343] examined the uptake, disposition, and effects of CCl_4 in rainbow trout after water exposure and after intraperitoneal injection. Injections of CCl_4 (1 ml/kg) produced 5- to 10-fold increases in serum glutamic oxalacetic transaminase, glutamic-pyruvic transaminase, and isocitric dehydrogenase, whereas exposure of trout to CCl_4 in tank water (1 to 80 mg/l) produced neither mortality nor significant changes in emzyme activites. Regardless of the route of administration, CCl_4 residue concentrations appeared highest in the adipose tissue, followed by liver, brain, and spleen, and were lowest in the gill. Elimination rates of ^{14}C residues from tissue samples were most rapid in muscle and most prolonged for the liver. Histological examination of tissues revealed varying degrees of liver and splenic necrosis 6 hours after injection of CCl_4.

Wales *et al.*[344] found that liver cancer was induced in rainbow trout eggs by exposure to an 0.5 mg/l solution of AFB_1 for 1 hour. The uptake, distribution, and elimination of ^{14}C-labelled AFB_1 in trout eggs was also described. Comparison of the amount of AFB_1 absorbed by trout eggs with the amount of AFB_1 contained in fish from an AFB_1 feeding study indicated that the trout embryo was even more sensitive than juvenile trout to the carcinogenic properties of AFB_1. The effect of a PCB (Aroclor 1254) on the hepatocarcinogenicity of AFB_1 in rainbow trout was determined by Hendricks *et al.*[345] After 12 months, 70.3% of the trout fed 6 μg AFB/l had hepatocellular carcinonas, compared to 30.4% of fish fed 6 μg AFB_1/l plus 100 mg/l Aroclor 1254. The concentration of Aroclor 1254, on a whole fish basis, increased rapidly for the first 6 months, then tended to equilibrate and plateau at approximately 80 mg/l for the remaining 6 months in fish fed diets containing 100 mg/l Aroclor 1254. AFB_1 inhibited growth of trout while Aroclor 1254 did not. The only changes induced by feeding Aroclor 1254 were glycogen depletion of hepatocytes, and white pulp depletion and hyperemia in the spleens. Brekke *et al.*[346] added either uncontaminated corn, aflatoxin-contaminated corn, ammoniated uncontaminated corn, or ammoniated aflatoxin-contaminated corn to the diet of rainbow trout and assessed the effects of ammoniation on the resulting carcinogenicity of these diets. Ammoniation inactivated the aflatoxins and reduced the carcinogenicity of the contaminated corn to a level that was not significantly different from that obtained with the basal control diet. The nutritive value of the ammoniated corn to trout appeared to be equal or slightly superior to that of the untreated corn. It was determined by Stott and Sinnhuber[347] that liver postmitochondrial fraction enzymes of rainbow trout were able to metabolize AFB_1 to an active mutagen to *Salmonella typhimurium*. The pretreatment of rainbow trout with various PCB compounds decreased the mutagen assay response to AFB_1. Stephenson *et al.*[348] subjected eggs, embryos, and fry of the Japanese medaka to different concentrations of AFB_1 and observed both toxic and teratogenic responses. The LC50 for eggs was 0.18 mg AFB_1/l over the 720-hour test period. Teratogenesis was noted for the circulatory system, optical development, appendage development, swim bladder, and spleen of exposed fish.

Hara and Thompson[349] found that whitefish (*Coregonus clupeaformis*) were attracted to sublethal concentrations of an anionic detergent, sodium lauryl sulfate (SLS). The fish were tested in a trough where a choice between clean and modified water was presented. The authors found that olfactory bulbar electrical responses to odorants were depressed when the nares were perfused with SLS and household detergents. The lowest concentration of SLS to cause minimum effects within 15 minutes was 0.1 mg/l. Avoidance reactions of young Ayu (*Plecoglossus altivelis*) exposed to various detergents were studied by Tatsukawa and Hidaka.[350] The estimated threshold concentrations of avoidance for Ayu were: 0.11 μg/l of formulation LAS, 1.5 μg/l of pure reagent LAS, 4.0 μg/l of formulation AS, 8.4 μg/l of pure reagent AS, 11.0 μg/l of pure reagent ABS, and 31.0 μg/l of soap. The activity of inorganic pyrophosphatase from muscle and liver tissues from detergent-exposed fish (*Misgurnus fossilis*) was studied by Niculescu and Mester.[351] *In vivo* and *in vitro* administration of sodium dodecylsulfate and sodium deoxycholate was found to inhibit pyrophosphatase activity in both tissues. According to Jackson and Fromm[352] perfused gills in rainbow trout exposed to 5, 10, 20, and 100 mg/l LAS responded with an exponential uptake of tritiated water during a 65-minute exposure. Mayer *et al.*[353] examined the synthesis of vertebral collagen and hydroxyproline in rainbow trout, brook trout, fathead minnows, and channel catfish after exposure to toxaphene, PCB (Aroclor 1254), the dimethylamine salt of 2,4-D (2,4-D DMA), and di-2-ethylhexyl phthalate (DEHP). Both collagen and hydroxyproline were decreased in fish exposed to toxaphene and Aroclor 1254. The collagen content of bone was significantly reduced in fish exposed to 2,4-D DMA or DEHP, but the hydroxyproline concentrations in collagen were either not affected or were significantly increased. The vertebral collagen content of the hydroxyproline concentration in collagen was discussed in relation to fish growth and to vitamin C content in the vertebrae of exposed fish. Hinz and Matsumura[354] compared the metabolism of ^{14}C-labelled 2,5,2′-trichlorobiphenyl observed in goldfish, rainbow trout, and bullheads (*Ictalurus* sp.) with the pattern of metabolism present in rats. There was little *in vitro* metabolism of 2,5,2′-trichlorobiphenyl by livers of rainbow trout and bullheads, whereas 6 to 9% of the 2,5,2′-trichlorobiphenyl incubated with goldfish liver enzymes was recovered as more polar metabolites. The *in*

vitro metabolic pattern of the goldfish was almost identical to that of the rat while the *in vivo* metabolic pattern was different. Effects of Clophen A50, 4-chlorobiphenyl (4-CB), 2,5,2′,5′-tetrachlorobiphenyl (TCB), 2,-4,5,2′,4′,5′-hexachlorobiphenyl (HCB) on the mixed-function oxidase system of rainbow trout liver were studied and compared by Förlin and Lidman.[355] Neither NADPH-cyt *c* reductase activity nor microsomal protein and phospholipid contents were affected by intraperitoneal injections of any of these compounds. Clophen A50 and HCB enhanced the o-demethylation of *p*-nitroanisole to *p*-nitrophenol and increased the content of cyt P-450 in the liver, while 4-CB and TCB had no effects of either of these parameters. The acute response of liver of channel catfish and rat to PCB was compared by Lipsky *et al.*[356] on both a structural and functional basis. Both catfish and rats had elevated microsomal enzyme activities after injection with PCB. Livers of treated catfish showed increased lipid accumulation and increased profiles of rough endoplasmic reticulum. Hacking *et al.*[357] determined the normal ultrastructure of the liver of rainbow trout and identified the modifications that occurred in this structure after chronic administration of PCB (Aroclor 1254). No ultrastructural liver changes were observed in fish fed diets containing 1 mg/l Aroclor 1254. The most common alterations observed in the 10 and 100 mg/l treated groups included irregular nuclear outlines, separation of nucleolar components, and large nuclear pseudoinclusions. Some histopathological effects of PCB (Aroclor 1254) injections on the liver and gonads of rainbow trout and carp (*Cyprinus carpio*) were reported by Sivarajah *et al.*[358] Aroclor 1254 produced general vacuolation of the hepatocytes of trout but not in carp. The ovaries of treated fish appeared to have fragmented cytoplasm but there were no apparent changes in the testis. In addition, enlargement of the rough endoplasmic reticulum of the hepatocytes, some damage to spermatozoa, and proliferation of the smooth endoplasmic reticulum of the oocytes of treated fish were observed. Sivarajah *et al.*[359] also examined the correlation between hepatic microsomal enzyme activities and changes in the steroid hormone levels in the blood of rainbow trout and carp after injection of Aroclor 1254. The activities of aminopyrine demethylase, p-nitroreductase, and UDP-glucuronyl-transferase were significantly increased in all treated fish. Cytochrome P-450 and microsomal protein contents were increased in treated trout but not in treated carp. There was a significant reduction in the plasma levels of androgens, oestrogens, and corticoids in the Aroclor 1254 treated fish, and there was a correlation between increased enzyme activities and a decrease in plasma hormone levels. Elcombe and Lech [360] examined two commercial PCB mixtures (Aroclor 1254 and Aroclor 1242) and one PBB mixture (FireMaster BP-6) for their abilities to induce hepatic microsomal monooxygenation in rainbow trout. Pretreatment of trout with Aroclors 1254 and 1242 (150 μg/g IP) or with FireMaster BP-6 (150 μg/g IP) resulted in an induction of arylhydrocarbon, (benzo [a] pyrene) hydroxylation, ethoxycoumarin-O-deethylation, and ethoxyresorufin-O-deethylation. Administration of these polyhalogenated biphenyls did not significantly affect P-450 hemoprotein or ethylmorphine-N-demethylation. Statham *et al.*[361] identified the effects of injections of 3-methylcholanthrene, 2,3-benzanthracene, and β-naphthoflavone on hepatic microsomal enzymes and on the disposition of 2-methylnaphthalene in rainbow trout. These three polycyclic aromatic hydrocarbons caused dramatic increases in hepatic arylhydrocarbon (Benzo [a] pyrene) hydroxylase activity and hepatic microsomal cytochrome P-450 levels without affecting glucose-6-phosphatase or glucuronyl transferase. Pretreatment of trout with 2,3-benzanthracene resulted in increased rates of metabolism and biliary excretion of 2-methylnaphthalene *in vivo*. Melancon *et al.*[362] investigated the effects of piperonyl butoxide on the disposition of DEHP by rainbow trout. Piperonyl butoxide (1 mg/l) *in vitro* inhibited the oxidation and hydrolysis of DEHP by liver homogenate fractions and serum from rainbow trout. Preexposure of trout to piperonyl butoxide *in vivo* modified the disposition and metabolism of DEHP *in vivo* and the major effects observed were decreased concentrations of phthalates in bile and increased concentrations in muscle. Liver toxicity in rainbow trout following injection with monochlorobenzene (CB) was evaluated by Gingerich and Dalich.[363] Slight inflammation of the peritoneal cavity and enlargement of the spleen occurred in several treated fish. CB injection caused elevated plasma GPT activity and reduced clearance of plasma sulfobromophthalein, both of which indicate that some hepatotoxicity was occurring in these trout. The median lethal dose of CB was estimated to be 1.8 ml/kg at 24 hours. Khvoles and Kor-

obko [364] exposed loach (*Misgurnus fossilis*) to dimethyl sulfate (DMS) for 6 hours and examined the effects on the reproductive system. DMS exposure not only increased the total number of nucleoli in the nucleus, but also increased the size of the nucleoli present. RNA concentrations in the nucleoli and cytoplasm of oocyte cells were also increased by DMS exposure. Daikoku [365] studied the effects of feeding trimethylamine (TMA) on the seawater adaption of guppies and goldfish. In both species, increasing the TMA contents of the diet increased the trimethylamine oxide (TMO) concentrations in muscle tissue of fishes in sea water; however, TMO concentrations of freshwater medium were higher than in the seawater medium. The effects of Bunker C oil and an oil dispersant (Oilsperse 43) on serum glucose levels, serum sodium levels, and gill morphology in both fresh and sea water acclimated rainbow trout were investigated by McKeown and March.[366] Both compounds tended to reduce serum glucose levels with Bunker C oil causing the greater decrease. Oilsperse 43 significantly lowered serum sodium concentrations in fresh water. Extensive deterioration of the gill structure resulted from exposure to Oilsperse 43 and a combination of Bunker C oil and Oilsperse 43 with less impairment resulting from exposure to Bunker C oil. The behavioral effects of acute bis (tri-n-butyltin) oxide (TBTO) exposure on rainbow trout and *Tilapia rendalli*, and the hematological and histological effects of TBTO exposure on rainbow trout were studied by Chliamovitch and Kuhn.[367] Based on the loss of positive rheotaxis, the 24-hour EC50 was 30.8 μg TBTO/l for trout and 53.2 μg TBTO/l for *T. rendalli*. Exposure to TBTO concentrations ranging from 0.023 to 1.17 mg/l increased the packed cell volume, the hemoglobin concentration, and erythrocyte count in rainbow trout. Histological damage caused by TBTO exposure was observed in the gills, liver, and eyes of rainbow trout. Smit *et al.*[368] compared heparin and EDTA as anticoagulants for the determination of pH, pCO_2, pO_2, and hematocrit values in the blood of carp. All concentrations of EDTA investigated caused changes in pH, pCO_2, pO_2, and hematocrit values of fish blood, whereas heparin produced few significant effects on these parameters.

Offutt and Gritzke [369] studied the influence of ethanol on the level of photic stimuli required to evoke an orienting heart rate response in goldfish. Goldfish exhibited orienting responses to less intense stimuli during the duration of all tests after ethanol was administered. There also appeared to be a residual increase in excitability lasting as long as 7 days after ethanol was administered. The alteration of thermoregulatory behavior in guppies and Atlantic salmon by 5-hydroxytryptamine (5-HT) was evaluated by Fryer and Ogilvie.[370] The lowest dose of 5-HT tested significantly decreased the selected temperature of guppies, while the highest dose significantly increased it. In salmon, injections of 5-HT initially decreased the selected temperature, although the selected temperature gradually increased, and by 3 hours after injection it exceeded the control value. Morton [371] found the intraperitoneal injection of sublethal doses of valinomycin, gramicidin D, and m-chlorocarbonyl-cyanidephenylhydrazone (CCP) reduced the heat resistance of goldfish. Possible mechanisms of action were suggested to explain the increased heat sensitivity of goldfish treated with these compounds. Tests were conducted by Kyono and Egami [372] to determine the effect of temperature during diethyl-nitrosamine (DENA) treatment on liver tumorigenesis in the medaka. Many large or small tumor nodules were found in all fish exposed to 100 mg/l DENA at a high temperature (25°C); at low temperature (8°C), the majority of fish had no nodules when treated with 100 mg/l. The histological changes observed in fish treated with DENA at low temperature appeared to be induced by the toxicity of the drug, and the step to tumorigenesis seemed to require an appropriate high temperature. In a similar study, Kyono [373] treated medaka with 100 mg/l of DENA for 8 weeks and examined temperature effects during and after treatment on liver tumorigenesis. Treated fish maintained continuously at high temperature (22°C) and the fish transferred to low temperature (5° to 6°C) after treatment at high temperature developed many tumor nodules in their livers, although the number and size of the nodules were larger in the group treated and held continuously at high temperature. Singley [374] investigated the hypophyseal-adrenocortical response to stress in the goldfish by observing changes that occurred in serum cortisol levels and ACTH titers after exposure to various stressing agents. The effects of prostaglandins, radiation, saline immersion, net capture, ice immobilization, MS-222 anesthesia, and electric immobilization on cortisol and ACTH levels were discussed.

INDUSTRIAL AND MUNICIPAL POLLUTANTS

Chlorine and Wastewater. Monthly 96-hour bioassays were carried out by Zietoun [375] to determine the effects of intermittent chlorination on the survival of caged rainbow trout (*Salmo gairdneri*) placed in a channel below an electric generating facility. The condenser chlorination at this plant occurred once daily for a period of 30 minutes. The results indicated that eight covariates, which included total and free chlorine exposure time integrations, DO supersaturation and temperature, contributed significantly to the mortalities observed among caged fish. A maximum value of 0.3 mg/l total residual chlorine per 54 minutes of exposure time was extrapolated as a safe concentration. Heath [376] simulated chlorine discharges from electro-generating plants by exposing rainbow trout, coho salmon (*Oncorhynchus kisutch*), golden shiner (*Notemigonus crysoleucas*), carp (*Cyprinus carpio*), and catfish (*Ictalurus punctatus*) to pulses of free chlorine or monochloramine three times a day for as long as 7 days under laboratory conditions. Temperature had little effect on the toxicity of intermittent chlorine to the species tested. It was found that free chlorine was 3 to 14 times more toxic, depending on the fish species tested, than monochloramine.

The acute toxicity of residual chlorine and ammonia was assayed by Roseboom and Richey [377] using bluegills (*Lepomis macrochirus*), channel catfish, and large-mouth bass (*Micropterus salmoides*). The 96-hour LC50 limits for residual chlorine were 0.18 to 0.33 mg/l for bluegills, depending on temperature and fish weight, and 0.09 mg/l for channel catfish with temperature not a factor. For ammonia the 96-hour LC50 limits were 0.40 to 1.3 mg/l for bluegills, depending on temperature and fish weight, 0.72 mg/l at 22°C to 0.5 mg/l at 30°C for bass, and 1.5 mg/l at 22°C to 3.0 mg/l at 28°C for channel catfish with size not a factor. Katz [378] demonstrated that the lethal toxicity of a given concentration of chlorine to adult *Gambusia affinis* was considerably reduced in the presence of various nitrogenous compounds. After determining the individual toxicities of chloramines, LAS, and Cu to goldfish (*Carassius auratus*), 96-hour LC50s of 0.35, 6.16, and 0.30 mg/l, respectively, Tsai and McKee [379] tested mixtures (two at a time) of these chemicals at various toxicity ratios and exposure periods. Mixtures of chloramines and LAS, chloramines and Cu, and LAS and Cu always exerted additive toxic action at the equal toxicity ratio of 1:1. When tested at unequal toxicity ratios, the results were either additive or synergistic, depending on the concentrations of the mixtures. Seegert and Brooks [380] exposed coho salmon, alewife (*Alosa pseudoharengus*), spottail shiner (*Notropic hudsonius*), and rainbow smelt (*Osmerus mordax*) to residual chlorine for 30 minutes at various temperatures. The 30-minute LC50 values were both species and temperature dependent. All species showed an inverse relationship between test temperatures and LC50 values (Table I). In a related study, Brooks and Seegert [381] tested 10 species of fish at 10°, 20°, and 30°C to determine their resistance to monochloramine administered every 5 hours for 40 minutes during a 24-hour period. The species, in decreasing order of sensitivity, were emerald shiner (*Notropis atherinoides*), spotfin shiner (*N. spilopterus*), common shiner (*N. cornutus*), channel catfish, white sucker (*Catostomus commersoni*), and sauger (*Stizostedion canadense*). The LC50 values for these species at 30°C ranged from 0.35 to 0.71 mg/l. More resistant species, freshwater drum (*Aplodinotus grunniens*), white bass (*Morone chrysops*), bluegill, and carp, had LC50 values of 1.15 to 1.50 mg/l at 30°C. Larson *et al.*[382] investigated the toxicity of chloramines to brook trout (*Salvelinus fontinalis*) alevin, fry, and juvenile life stages. Although toxicity was not constant between the life stages, the life stage body weight relationship did have a measurable influence on the 96-hour LC50 values (Table I). Another study by Larson *et al.*[383] assessed the acute and chronic toxicity and behavioral effects of chlorine and inorganic chloramines on several species of fish. The acute toxicities of inorganic chloramines were less than 100 μg/l for salmonids and were a function of life-history stage and some water quality conditions. Values between 10 and 22 μg/l caused adverse effects on growth of alevins and juveniles. Behavioral responses of fish, such as avoidance and lethargic swimming and surfacing occurred not only at acutely toxic concentrations but also at lower ones. In addition, stabilized pulp mill effluents, which were not usually acutely toxic, became acutely toxic after chlorination even though no residual chlorine was detectable in the test solutions. Ward and DeGraeve [384] demonstrated that mean concentrations as low as 0.033 mg/l residual chlorine and 0.034 mg/l residual bromide chloride retarded the

growth of fathead minnows (*Pimephales promelas*). A mean residual ozone concentration of 0.016 mg/l or less did not affect survival growth, or reproduction of this species. Fava and Tsai [385] found that blacknose dace (*Rhinichthys atratulus*) avoided concentrations of 0.07 and 0.17 mg/l total chlorine (chloramines) and 0.07, 0.21, and 0.47 mg/l free chlorine. Fish exhibited the capability to avoid both chloramines and free chlorine in water before receiving a lethal dose. The avoidance reaction of blacknose dace to free chlorine had a longer latent period and median avoidance time than to chloramines at the same total chlorine concentration. A rise in blood viscosity among rainbow trout exposed to chlorine was attributed to an increase in both plasma protein concentration and hematocrit (Zeitoun [386]). Hemoconcentration of the blood was accompanied by a significant increase of the methemoglobin and hemoglobin concentration. Hemoconcentration also seemed to interfere with blood circulation and delivery of O_2 to the tissues. Lauver and Sellers [387] noted that additional blood plasma proteins appeared in rainbow trout exposed to 0.073 to 0.22 mg/l chlorine. Brown *et al.*[388] studied the effects of various disinfectants on the recovery of polio viruses from fathead minnows grown for 11 months in treated wastewater. Polio viruses, types 1 to 3, were present in the intestinal tracts of the fish raised in nondisinfected wastewater and in those exposed to wastewater treated with bromochloride and ozone. These viruses were not present in the intestines of fish raised in wastewater treated with chlorine. Mukherjee and Bhattacharya [389] exposed *Ophicephalus punctatus* and *Clarias batrachus* to a mixture of industrial effluents, sodium sulfide, phenol, ammonia, and copper sulfate and found that all caused significant inhibition of enzyme activities, even at low sublethal doses.

Starnes [390] studied the fish and aquatic invertebrates from two branches (Prongs) of the Little Pigeon River (Tennessee) to gain insight into possible changes in their populations due to pollution by municipal effluents. A reduction in the total numbers and the number of species of Percidae was found in the polluted West Prong, below the resort city of Gatlinburg, compared to the more pristine Middle Prong. In comparing the fauna of two rivers of similar geologic background, Zunjic [391] found that several species of salmonids had disappeared from the one river receiving cellulose industrial effluents. Ward *et al.*[392] selected an activated sludge, trickling filter treated effluent composed of 35 to 45% industrial wastes to determine what effects various disinfection procedures had on the survival of both cold- and warm-water fishes and the life cycle of fathead minnows. Nondisinfected effluent was inherently toxic to most species of fish tested. Salmonid fish were more sensitive to chlorobrominated effluent than to any other effluent type. The chlorinated effluent was the most toxic effluent type tested. Dechlorination with sulfur dioxide reduced the toxicity of the chlorinated effluent to levels similar to the nondisinfected effluent. Adverse effects on the growth and reproduction (life cycle tests) of fathead minnows were not attributed to any of the disinfectants tested. In a similar study, Ward and DeGraeve [393] conducted acute toxicity tests associated with chlorinated wastewater that had been dechlorinated with sulfur dioxide. The chlorinated effluent was acutely toxic to cyprinid, salmonid, and centrachid fishes. LC50 values ranged from 0.045 to 0.278 mg/l as total residual chlorine. Residual bromine chloride was generally less toxic than residual chlorine. In tests with ozonated effluent, LC50 concentrations could not be calculated due to lack of mortality or because animals were exposed only to 100% effluent. The acute residual toxicity of chlorinated effluent was eliminated by dechlorination with sulfur dioxide. In a study of a complex wastewater treatment plant by EPA,[394] water-fleas (*Daphnia magna*), algae (*Selenastrum caprincornutum*), and bluegill sunfish were exposed to various concentrations of the plants' influent and effluent. All were toxic to water-fleas and bluegills but not toxic to algae. The wastewater stimulated algal growth. Servizi *et al.*[395] conducted flow-through and static bioassays of a dechlorinated primary wastewater treatment plant effluent. Based on tests using fingerling sockeye salmon (*Oncorhynchus nerka*), acute toxicity was greater during flow-through tests than during static bioassays. Acute toxicity also was greater during dry weather than during wet weather flow conditions. Alexander *et al.*[396] found that most species of fish and invertebrates exposed to a nondisinfected domestic effluent survived at concentrations less than 100%. Invertebrates were more sensitive than Artic char (*Salvelinus alpinus*), lake trout (*S. namaycush*), rainbow trout, and whitefish (*Coregonus clupeaformis*). Rainbow trout were more sensitive in static tests than flow-through tests and when forced to swim were

more susceptible to wastewater than passively exposed fish. In avoidance tests, whitefish actually preferred some effluent concentrations and did not avoid any effluent concentration tested. Rawlings [397] reported on the first phase of a study designed to provide chemical and toxicological baseline data on wastewater samples collected from textile plants in the U. S. Various bioassay tests were performed using secondary effluent samples collected from 23 plants. Fathead minnows were used as the test animal in some of these bioassays. Overall bioassay results indicated that 10 of the 23 plants have secondary effluent sufficiently toxic to warrant further study. Phase two of this study, involving these 10 plants, was designed to determine the level of toxicity removed by selective tertiary treatment technologies. Trivedi and Dubey [398] measured 24- and 48-hour toxicities of effluents from viscose rayon/caustic soda industries to the river fish (*Barbus stigma*) found in India. The main pollutants present in these effluents were determined to be Zn, Hg, sulfuric acid, and organic matter. Wastes diluted as much as 40 times were still found to be lethal in 48-hour tests. Acute values for wastewater containing presoaking, soaking, liming, reliming, deliming, bating, pickling, chrome-tanning neutralization, retanning and dyeing-fat-liquoring of leather manufacturing processes were measured using cyprinodonts as test fish.[399] Of these processes studied, the deliming/bating and retanning process yielded the highest LC50 values. Prazakova [400] noted that wastewater from a poultry slaughter plant and town wastewater was suitable for use in raising carp in fish ponds. The feasibility of using reclaimed municipal wastewater as makeup water for recreational lakes, concentrating mainly on water quality in relation to fish maintenance, algal growth and control, and phosphorus levels was examined by Headstream *et al.*[401] Ponds fed with recovered percolated effluent supported numbers of channel catfish and fingerling bass.

With the aid of a computer, Van der Schalie [402] recorded the ventilatory patterns of bluegill sunfish to monitor the toxicity of an industrial effluent. No known toxic spills occurred in the effluent during any experiment. Acetone added to the effluent (simulated spill) caused responses from the fish at concentrations that peaked near the 96-hour LC50 level. The design and cost of future monitoring units similar to the one tested were discussed. Carlson and Drummond [403] exposed bluegill sunfish for 24 hours to a variety of industrial effluents. They found cough frequency increased commensurate with the effluent concentration and concluded that the response was a rapid and sensitive measure of effluent quality. Lésel and Huez [404] reported that swimming resistance of rainbow trout was directly related, depending on the condition of the effluent, to the concentration of a crude tannery effluent.

Pulp and Paper. Growth and food selection of native larval fish were not influenced by a pulp mill discharge according to Leslie and Kelso.[405] They concluded that either the larval fish were unaffected by the mill effluent or the captures in the effluent discharge area were transients. Juul and Shireman [406] surveyed naturally occurring fish populations within a kraft pulp mill effluent channel. Although reproduction was not observed for any species, except mosquitofish (*Gambusia affinis*), the authors reported water quality remained favorable for fish survival and growth throughout the study period. As an example, growth rates of white catfish (*Ictalurus catus*) and warmouth (*Lepomis gulosus*) appeared to be greater than or equal to growth rates reported for these species in other natural waters. Nyronen [407] studied the age-class distribution and growth rates of fish in a lake in Denmark polluted mainly by organic matter from pulp mills. The relation of the changes in the fish populations, based on tests from the years 1968, 1973, and 1975, with the changes of pulp mill effluents were discussed.

The toxicity of pulp mill wastes and pure compounds isolated from these wastes were investigated by Leach and Thakore.[408] Major toxic factors in effluents from Canadian softwood kraft, sulfite and mechanical pulping operations, and wood debarking effluents were seven resin acids: dehydroabietic, abietic, isopimaric, palustric, pimaric, sandaracopimaric, and neoabietic. The 96-hour LC50 values of the pure compounds for coho salmon (*Oncorhynchus kisutch*) ranged from 0.2 to 0.8 mg/l. In tests with caustic extraction effluents using rainbow trout (*Salmo gairdneri*), the predominant toxic contributors were trichloro- and tetrachloro-guaiacol, monochloro- and dichlorodehydroabietic acid, (96-hour LC50, 0.3 to 0.75 mg/l), and 9,10 epoxy and 9,10 dichlorostearic acid (96-hour LC50, 1.5 and 2.5 mg/l, respectively). These and some other toxicants present in these wastes also were discussed in relation to their source and effluent treatment techniques. Fox *et al.*[409] examined rainbow trout for the presence of dehydroabietic acid

after they were exposed to 3 to 18% kraft mill effluent for 48 to 144 hours. This acid was chosen because of its demonstrated toxicity and known persistence in both waters and sediments. Levels of 2 to 10 μg/g dehydroabietic acid were found based on a whole fish wet weight. They concluded that fish exposed to kraft mill effluent accumulated dehydroabietic acid to a level that may cause sublethal toxic effects. Göttsching and Geller [410] evaluated the use of the golden orfe (*Leuciscus idus melanotus*) to assess the acute toxicity of pulp and paper industry effluents. Some of the legal considerations and bioassay procedures that appear to be problem areas were discussed. Werner and Robinson [411] found that concentrations of pulp fiber suspensions exceeding 1 000 mg/l were lethal to sockeye salmon fingerlings (*Oncorhynchus nerka*) tested in fresh water. Even the most highly purified fibers (acetic acid-washed acetone extracted, kraft) possess a residual acute lethality due to the fibers themselves. The acute toxicity of lignosulfonates (sodium lignin sulfonate) found especially in sulfite pulp mill effluents was investigated by Roald.[412] The 48-hour LC50 value for lignosulfonates for rainbow trout was calculated to be 7 300 mg/l. During a 28-day exposure, 100% survival was recorded at concentrations of lignosulfonates as high as 1 875 mg/l. Another study by Roald [413] showed that the growth rate of young rainbow trout exposed to concentrations of 160 mg/l lignosulfonate and higher was slower than that of controls. Lignosulfonates had no major influence on the composition of intestinal bacterial flora in trout, but a significant reduction of enzyme activities in three of the four groups of digestive enzymes tested was observed. After showing that herring, *Clupea harengus*, avoided a sulfite pulp mill effluent, Wildish *et al.*[414] suggested that herring may avoid long chain complex molecules such as lignosulfonates rather than low molecular weight compounds in pulp mill effluents. McLeay and Gordon [415] measured leucocrit (volume of packed leucocytes-thrombocytes expressed as a percentage of whole blood) values for rainbow trout and coho salmon exposed to a bleach kraft mill effluent. Leucocrit values were depressed when the fish were subjected to conditions of high temperature, crowding, transfer stress, and exposure to acute concentrations of the effluent. Toxicity and behavioral bioassays were conducted by Eedy and Schiefer [416] with a simulated kraft pulp mill effluent using landlocked Atlantic salmon (*Salmo salar*), anadromous Atlantic salmon, and rainbow trout. All fish were able to survive 96 hours in up to 80% of the effluent. In some behavioral tests, using the landlocked salmon and rainbow trout, no preference or avoidance reactions were indicated at effluent concentrations of 5% or less. At a concentration of 10%, they avoided the effluent 70% of the time. Total avoidance was never observed even at concentrations greater than 25%.

Radioactivity. Ijira *et al.*[447] irradiated fish (*Oryzias latipes*) with X-rays using dose rates of 2 800, 400, 100 and 20 R/min. A single wholebody dose of 80 kR or less caused no mortality within 24 hours of irradiation, whereas doses of 90 kR or more were lethal to some fish. Aleksanyan *et al.*[418] determined the toxic effect of ^{90}Sr and metaphos on biochemical and physiological characteristics of carp (*Cyprinus carpio*). The combined action of the radio-nuclide and pesticide was found to produce a less toxic action on the fish than each taken separately.

The relative rate of radioactive manganese (^{54}Mn) uptake by fathead minnows (*Pimephales promelas*) was investigated by Kwasnik *et al.*[419] The fish were exposed to activity levels ranging from 5.5 to 8.5 μCi ^{54}Mn/l. The maximum accumulation of ^{54}Mn in fathead minnows was reached at 128 hours. The uptake of ^{54}Mn showed a transient decrease in activity after 4 hours of exposure, following an initial increase in activity. Till [420] conducted a study that focused on the uptake and distribution of ^{238}Pu(IV) citrate by carp eggs and the toxicity of Pu to both carp and fathead minnow eggs. Autoradiographs of carp eggs revealed, although some Pu was concentrated on the egg chorion, that Pu that penetrated the chorion was uniformly distributed throughout the perivitelline fluid, embryo, and yolk sac. Concentrations of 1 mCi/l were required to prevent both species of eggs from hatching. Eyman and Trabalka [421] administered, by intragastric injection, three chemical forms of ^{237}Pu (Pu-citrate, Pu-fulvate, and Pu-hydroxide) to channel catfish (*Ictalurus punctatus*). They found that chelation can either enhance or reduce uptake of injected Pu relative to PuOH (monomer) in catfish. Reduced uptake of ^{237}Pu-fulvate was due to molecular weight of the complex or its stability in metabolic systems. Increased uptake of ^{237}Pu-citrate was attributed to the instability of the complex of metabolic systems. Kimura and Honda [422] studied the uptake and elimination of radionuclides, ^{60}Co, ^{131}I, ^{137}Cs, ^{144}Ce, and

^{106}Ru, by fertilized eggs of rainbow trout (*Salmo gairdneri*) and found uptake and accumulation of ^{60}Co and ^{137}Cs were more rapid than those of the other radionuclides. In terms of elimination, ^{106}Ru was much slower compared to the others studied. The significant fraction of ^{60}Co, ^{131}I, ^{137}Cs, and ^{144}Ce in the egg was associated with the perivitelline fluid, while the greatest part of ^{106}Ru in the egg was accumulated in the egg capsule. These authors [423] also studied the uptake and elimination rates, using these same radionuclides, by fry and fingerling rainbow trout held under laboratory conditions. Tests to determine the distribution pattern of tritium in the body water and edible tissues of *Tilapia mossambica* exposed to a medium concentration of 0.04 mCi/l were conducted by Krishnamoorthy *et al.*[424] After 30 days of exposure, the tissue-free water tritium equilibrated rapidly to 97% of the medium concentration while the tissue-bound tritium reached only 3.5% of the tissue-free water tritium. Strand *et al.*[425] intraperitoneally injected a heat-killed strain of *Flexibacter columnaris* to investigate the effect of tritium irradiation (0, 0.04, 0.4, 4.0, 40 rads total dose for 20 days during embryogenesis) on development of the primary immune response in 5-month-old rainbow trout, reared under essentially pathogen-free conditions. It was found that agglutinin levels in irradiated fish were suppressed 50% of control levels at 40 rads during the ninth weeks, and 50% of control levels at 4.0 rads during the eleventh week. Of four major serum protein fractions (electrophoretic separations), the relative percent of total protein contained in fraction IV was found to be reduced in irradiated fish. Mărgineanu and Chiosila [426] developed a mathematical frame for the analysis of experimental data concerning the incorporation of ^{85}Sr in goldfish (*Carassius auratus*). The experimental curves giving the time course of radioactivity in fish and water were compared to the theoretical ones. On this basis, the water volume from which ^{85}Sr is retained by the fish was estimated at 0.3 to 0.5 cm^3/g·d.

Chinook salmon (*Oncorhynchus tshawytscha*) eggs and alevins were exposed by Hershberger *et al.*[427] to various levels of continuous gamma irradiation to determine the level at which the return of these individuals as adults would be affected. The dose rates ranged from 0.5 R/d to 50 R/d. After rearing for about 90 days, the control and irradiated groups were differentially marked and released for their migration to the sea. On their return 2 to 6 years later, no significant change was noted in adult returns until irradiation levels of 10 R/d and above were used. At these levels (10 R/d, 20 R/d, and 17 to 50 R/d), analyses showed retardation in growth and increased mortality of the small salmon in fresh water, decreased numbers of adults returning to spawn, increased age at return, and apparent sterility in the adult males. Spieser and Schröder [428] studied the response of substrains of male guppies (*Poecilia reticulata*) irradiated (1 000 R X-rays) in fresh and sea water. Four behavioral patterns were chosen for study: approaching, penetrating, sigmoidal position, and gonopodial swinging. The authors found mean values of both the frequency and the duration of the four behavioral traits increased in post-irradiation F_1 of the seawater substrain, but these traits were not changed in post-irradiation F_1 of freshwater guppies. In post-irradiation F_2, however, the mean values of the same behavioral characters decreased in both sea and fresh water substrains.

Other. Maki and Macek [429] reported that 680 mg/l suspensions of Type-A zeolite, a detergent builder, were not toxic to bluegills (*Lepomis macrochirus*), channel catfish (*Ictalurus punctatus*), and rainbow trout (*Salmo gairdneri*). The MATC for fathead minnows (*Pimephales promelas*) was greater than 87 mg/l. Hamburger *et al.*[430] exposed minnows (*Phoxinus laevis*), rainbow trout, and golden orfe (*Leuciscus idus*) to three dyestuffs, three auxiliaries, and one heavy metal salt. Results indicated that minnows, trout, and golden orfe were fairly equal in sensitivity to these seven types of chemicals. Schultz *et al.*[431] examined the toxicity of gasifier condensate water (Synthane process) to fathead minnows tested at 18°C. According to these authors, a more accurate estimate of the incipient LC50 was made using 7-day rather than 96-hour toxicity data. When percent mortality at 7 days was plotted against the concentration using probit-log transformation, the incipient LC50 was calculated to be 0.012% condensate. Prater and Anderson [432] collected sediment samples reflecting harbor areas that were suspected of being nonpolluted, moderately polluted, or heavily polluted. Using a recirculating test water system, 96-hour sediment bioassays were conducted with fathead minnows and other aquatic organisms. Sediment samples considered moderately to heavily polluted caused only a 10% mortality among fathead minnows. Other test organisms were found to be much more sensitive. Halsband [433] stated that 90%

of the roach collected from a German river, having a high concentration of potash, had severe ulcers. Other fish species also were being affected based on hematological studies.

PHYSICAL FACTORS

Galloway and Cowling [434] described the changing chemistry of precipitation and its effects on terrestrial and aquatic ecosystems, explained the concept of sensitive areas, and discussed the need for a network of precipitation measuring stations in the United States and Canada. Potential effects of chemicals include inputs of N, P, and K (fertilization) or H^+, pesticides, and toxic metal ions into the aquatic ecosystem. The authors found that certain aquatic systems were more sensitive to decreases in precipitation pH than others, resulting in conditions that would not sustain fish populations. Mitsch *et al.*[435] calculated the total energy impact of drainage on an ecosystem from biomass measurements, upstream and downstream species identification of benthic invertebrates and fish, application of a P/B ratio for the populations, and an estimate of the reach affected by drainage from an abandoned coal mine. The authors suggested that the impact of pollution on ecosystems can be summarized by determining the energetic cost to the ecosystem. White *et al.*[436] found extensive and possibly enhanced fish populations in a small spring-fed stream beginning to show the influence of urban development along its banks. Although sedimentation and discharge from wastewater treatment plants were beginning to affect the distribution of fish, a total of 63 species were collected, including some that inhabit small streams and some from the Ohio River that used the stream as a breeding and foraging ground. Short *et al.*[437] surveyed macroinvertebrates, periphyton, and fishes from a small stream flowing through an area subject to alterations due to road building, home development, and reservoir construction. Brook trout (*Salvelinus fontinalis*), the dominant species at an upstream control site, were somewhat depressed in numbers near the reservoir, but increased at lower sites. Although community composition between sampling sites differed and suggested that conditions have changed in some way, index values indicated no significant degradation of water quality below the reservoir. Koblitskaya [438] discussed succession of spawning communities in the Volga delta that had occurred over the past 30 years and considered the effect on them after a natural environmental disturbance when the Caspian Sea level dropped sharply in 1930 to 1941. The author suggested that the regulation of the Volga discharge had upset natural processes considerably and would accelerate succession, particularly in young communities, when a water divider was placed in operation. Contreras Balderas *et al.*[439] reported that 23 out of 27 areas surveyed in northern Mexico had fish communities that decreased by 16.7 to 100% due to adverse factors, which included damming, water over-exploitation, and salinization (as a consequence of dams). According to Cuinat,[440] the principal factors threatening the survival of salmon (*Salmo salar*) in the Loire-Allier streams included various types of pollution, dam construction, working of gravel pits, and overfishing, all of which had contributed to a reduction in stocks. Only about 60 km had acceptable conditions for survival of eggs and young fish. The effects of potato farming and clearcut logging upon fish, benthos, and physical characteristics of 33 surveyed streams in western New Brunswick were determined by Welch *et al.*[441] Clearcut logged streams had 17% fewer trout 200% more sculpins, and 26% less benthos than control streams, while farmed watershed streams contained 52% fewer trout, 92% fewer sculpins, and 64% less benthos compared with the control. Sedimentation was a contributing factor in farmed watershed streams, but low numbers of fish and benthos in these streams were mostly associated with chemical contamination. The effects of man's activities and the indirect influences of riparian vegetation on salmonid habitat were discussed by Meehan *et al.*[442] Influential factors of riparian vegetation included shade and organic detritus, which controlled the food base of the stream, and large woody debris that affected channel morphology.

Electrofishing surveys completed by Edwards [443] showed that most river fish populations were detrimentally affected by conventional channelization and channelization with mitigation when compared to a natural segment of the river. The mitigated area maintained an inferior community structure but was more successful in ameliorating recovery and reducing losses compared to the conventionally channeled segment. Barnard, [444] in a report devoted primarily to the morphometric and morphologic changes occurring in streams after channelization, discussed briefly the impact these may have on wildlife. The author found that channelization often transformed a meandering stream with tranquil pools and short, active riffles (flow conditions generally re-

quired by fish and other stream life) into a straight stream with numerous long riffles, few pools, and steeper gradients. A survey of fish populations by Carroll *et al.*[445] did not reveal significant differences in species diversity indices between channelized and unchannelized stretches of the South Sulphur River, Tex. Thirteen of the 15 species of fish collected from unchannelized stretches were collected from channelized areas, the exceptions being the carp (*Cyprinus carpio*) and blackstripe topminnow (*Fundulus notatus*). However, there was a significant difference in the index between values found upstream compared to those found downstream from a wastewater effluent discharged into the river. Porter [446] evaluated differences in macroinvertebrate and fish populations between channelized and unchannelized stream sections and found more favorable conditions for fish populations in the unchannelized area. Visibly greater fish habitat existed in the unchannelized section and more fish and fish species were found in unchannelized samples. Also, potential fish food was reduced in the channelized section because of fewer terrestrial drifters and more macroinvertebrate burrowers.

Paragamian [447] documented studies on the development of fish populations following impoundment in two flood control reservoirs. Fish species suited to river type habitat developed more extensively in one reservoir, which was initially dominated by carp and carpsucker, while crappie and carp became predominant in the other reservoir. Generally, growth of most species was best during the first few years of impoundment; and in the later years, growth was influenced by storage volume and water quality. Mayhew [448] identified factors related to flood control in a storage and conservation reservoir that influenced larval fish and zooplankton abundance, distribution, and survival. Flushing rate was the most important parameter. Larval gizzard shad (*Dorosoma cepedianum*) population density increased linearly with increased discharge outflow and lower flushing rate during seasons of increased flood control. A report by Walburg [449] described changes in fish population occurring in Lake Francis Case, one of six main stem reservoirs constructed on the upper Missouri River, during 23 years of impoundment. It was suggested that changes in abundance and species composition resulted from poor reproductive success due to water level fluctuation during spring and early summer, along with physical changes in the shoreline environment. Because fish reproduction was most successful in years when the reservoir was filled in spring and a nearly constant level maintained through July, it was recommended that a 3-year cycle in pool level management be instituted. Nelson and Walburg [450] described the development of yellow perch (*Perca flavescens*), sauger (*Stizostedion canadense*), and walleye (*Stizostedion vitreum*) in four main stem Missouri River reservoirs. Saugers, the predominant river percid, declined after impoundment due to loss of river spawning habitat and increased water clarity; abundance of yellow perch, low in the original river, increased as the reservoirs filled. However, these later declined and walleye populations slowly developed and became well established. Reproductive success was the primary factor regulating percid abundance. Blake [451] analyzed numbers and species composition of the Mormyridae since impoundment of the Niger River and formation of Lake Kainju (Nigeria). These fish, once diverse and commercially abundant in the relevant areas of the Niger, declined after impoundment but have since increased. Increases, however, were restricted to a few species only while many forms common in the river declined or have disappeared altogether from the lake. Further trends in the changing fish population between 1974 and 1975 in Lake Kainji were assessed and compared with pre- and post-impoundment data collected by previous workers.[452] The cichlids, particularly *Sarotherodou galilaeus*, became the dominant commercially available fish. Also abundant, but commercially underexploited, were the Shilbeidae. Holcik [453] analyzed changes in fish stocks, with special emphasis on the Eurasian perch (*Perca fluviatilis*), that occurred after filling the Klicava Reservoir (Czechoslovakia) and concluded that population changes were analogous to those in other, similar man-made lakes. Changes caused by impoundment in temperature, chemistry, DO, and the discharge regime of the River Tees immediately downstream from Cow Green reservoir were examined by Crisp.[454] The data showed that the annual temperature fluctuations dropped in amplitude by 1° to 2°C, diel temperature fluctuations were reduced, spring rise in temperature was delayed 20 to 50 days, and autumn and fall by 0 to 20 days. These temperature changes reduced the annual ration of 10 g trout by 15% and delayed the annual metabolic peak by 1 month. Walker *et al.*[455] reported that several native fish species have been displaced from part of their former range

in the Murray River (Australia) since dam construction affected significant downstream changes. These included higher-than-normal flows over the dry summer, reduction in amplitude of the seasonal temperature cycle by about 6°C, and a 1-month delay in spring, summer, and fall river temperatures. The frequency of sport fish attracted to artificial reefs placed in a reservoir generally coincided with their overall abundance.[456] Sunfishes (*Lepomis* sp.) and white catfish (*Ictalurus catus*) were the most common, but centrarchid bases were also well represented in the reef community. These results indicated that artificial reefs placed in structure deficient freshwater lentic environments were a viable fisheries management tool.

Hall [457] discussed approaches to the study of effects of watershed practices on stream fish populations in Oregon, including a 15-year study of three watersheds before and after logging, an intensive study of a logged and unlogged section in one stream several years after completion of logging, and a short-term study of several watersheds 1 year before and 1 year after logging. Also discussed were the physical and biological characteristics of a logged stream that could have contributed to significant reductions in resident cutthroat trout (*Salmo clarki*) populations, while leaving coho salmon (*Oncorhynchus kisutch*) and anadromous cutthroat populations relatively unaffected. Mortensen [458] assessed the impact of weed cutting and cleaning of the stream bed on the mortality rate of trout fry. Above an initial density of 3.1 fry m^{-2}, natural mortality rate was density-dependent in the first few months of life, but in stream reaches where weed cutting and cleaning had taken place, mortality rate was higher than that under normal conditions. Findings of the Aquatic Disposal Field Investigations (ADFI), conducted under the Dredged Material Research Program to provide definitive information on the environmental impact of dredging and disposal operations as well as advice as to how undesirable impacts could be reduced or eliminated, were summarized by Wright.[459] At the only freshwater site ADFI studied, adult and young pelagic fish were unaffected by disposal of sand and silt sediment into Lake Erie, although some bottom-dwelling fish migrated temporarily from the disposal area. Heavy metals in fish and invertebrates showed little change as a result of disposal. Carline and Brynildson [460] described the effects of hydraulic dredging on physiochemical characteristics of spring ponds, and particularly on changes in trout populations. In two ponds, studied for 2 years before and 4 to 5 years after dredging, volumes increased 400 to 500% and trout biomass nearly tripled from 36 to 90 kg/ha in one pond, but the standing crop of brook trout changed little in the other. Brown trout (*Salmo trutta*) accounted for more than half of the biomass increase in the first pond. Emigration was much reduced after dredging, and growth rates declined, but were within the predredging range within 4 to 5 years. Changes in growth rates were associated with fluctuations in densities of benthic organisms, the primary food of trout, which declined sharply during dredging but rapidly recolonized thereafter. In addition to reporting the results of a study designed to monitor in detail a small, hardwater, brook trout stream before, during, and after highway construction, Mathers [461, 462] assessed the effectiveness of sediment control techniques used during construction. Brook trout biomass, 107 kg/ha before construction, fell to 27 mg/ha and total fish biomass fell to 174 to 47 kg/ha during construction, whereas invertebrate species composition and biomass remained about the same downstream of construction.

Assessing the effects of granitic bedload sediment on juvenile salmonids and aquatic insects, Bjornn *et al.*[463] found that, during both winter and summer, fewer fish remained in artificial stream channels where sediment was added to the pools, fish numbers proportionately decreased to the loss in pool volume when sediment was added to small natural pools (100 to 200 m^2), and survival and emergence of salmonid embryos declined where fine sediment exceeded 20 to 30% in spawning riffles. However, significant correlations between riffle sedimentation and fish density were not observed in two natural streams studied. Meehan and Swanston [464] reported slightly higher survival of pink salmon (*Oncorhynchus gorbuscha*) eggs incubated in angular gravel than in other gravel types even though angular gravels accumulated somewhat more sediment during a range of waterflow conditions. Under constant flow conditions, round gravel accumulated more sediment during low flows (< 0.2 m^3/s) than other gravels, and at higher discharges (> 0.4 m^3/s), angular gravel accumulated more sediment. Samples of taconite tailings slurry demonstrated no toxicity in 22- to 49-day tests conducted by Oxberry *et al.*[465] with juvenile salmonids and perch (*Perca flavescens*), in 84- to 119-day tests with salmonids and embryos and larvae, and in 98- to

363-day tests with Lake Superior invertebrates. Clarified, partially neutralized extracts made by suspending tailings in acidified water (pH 5.0 to 5.5) were acutely toxic to rainbow trout (*Salmo gairdneri*). However, if the pH were raised from 5.0 to 6.9 one day before testing, the trout were not visibly affected, and it was concluded that the slightly alkaline Lake Superior waters would not become toxic to fish by dissolution of taconite tailings settling to the lake bottom. Peddicord and McFarland [466] evaluated the impact of sediment suspensions on marine, estuarine, and freshwater fish and invertebrates and concluded that suspensions of contaminated sediment were potentially more harmful than uncontaminated. Except for fluid muds, suspended sediment conditions created in the water column by most dredging or disposal operations were unlikely to cause ecological degradation. In freshwater experiments, golden shiners (*Notemigonus crysoleucas*) and fingerling rainbow trout were very tolerant to the suspensions. Using an innovative computer program to calculate diversity indexes, Reed [467] investigated aquatic macrobenthic and fish community responses to siltation effects from highway construction. Increased siltation and sedimentations resulted in a 20% reduction in numbers of fish species and a 40% drop in density, but fish were able to repopulate vacated areas within 12 months after the end of construction, depending on the stream's cleansing ability. In general, the study determined that the commonly applied erosion control measures used in highway construction were of limited value in preventing damages to stream communities. Alexander and Hansen [468] assessed the impact of greatly increased sediment in two trout streams caused by a gas-oil well drilling accident and reported population decreases for both spring and fall trout in the most affected stream but no measurable change in trout stocks of the lesser affected stream. Dissolved solids and DO concentrations along with temperature remained normal following the accident, but sediment concentrations peaked at 4 600 mg/l shortly thereafter in the most affected stream and declined to 100 mg/l or less within 2 weeks, compared to normal concentrations of around 15 mg/l sediment. In a study on feeding behavior of walleye and sauger, Swenson [469] reported that red-clay turbidity and inshore walleye density in Lake Superior were positively correlated.

The acute effects of underwater construction blasting on caged fish, reported by Teleki and Chamberlain,[470] included swimbladder ruptures and hemorrhaging in the coelomic and pericardial cavities. Fatality radii ranged from 20 to 50 m for a 22.7 kg charge of high explosive to 45 to 110 m for a 272.4 kg charge in 4 to 8 m of water. The blast gradient affected laterally compressed species more than the fusiform fishes. Of free-swimming fish, 47% of the total blast mortality was not visible from the water surface, and surface mortalities were high only in spring and late summer. Casillas and Smith [471] studied the effects of "hook and line" stress on blood coagulation and hematology in both a hatchery strain and wild type rainbow trout. Although the wild type recovered more quickly from the stress, blood clotting times declined and thrombocyte counts, hematocrits, and blood glucose levels increased in both strains. These results implied that an activated blood coagulation system in fish may be a sensitive indicator of sublethal environmental stress. Pickering and Macy [472] studied the structure, histochemistry, and effect of handling stress on the epidermis of char (*Salvelinus alpinus*). A single incidence of handling increased significantly the mucous cell concentration in the epidermis but, even with repeated handling, the effect was maximal after 1 week and was reduced to control levels after 1 month. In his thesis, Strange [473] reported changes in plasma cortisol concentrations over time in juvenile salmonids subjected to various stressors, including handling, confinement, temperature change, anesthetization, and introduction of sea water. Netted and confined chinook salmon (*Oncorhynchus tshawytscha*) showed an increase in plasma cortisol from 100 μg/l to about 500 μg/l in 24 hours, while in severely confined fish, it rose to 400 μg/l within 1.5 hours. Plasma cortisol was comparable to that of nonanesthetized controls in yearling chinook salmon exposed to 50 μg MS-222/l during mild handling, but anesthetic administered before initial capture effectively increased survival if a second stressor was applied. Cairns and Christian [474] determined the effects of hemorrhagic stress, caused by frequent and repeated blood sampling, on blood parameters of adult rainbow trout. Hematocrit, lactic dehydrogenase, and creatine phosphokinase were the most significant indicators of stress during the sequential, daily (7 days), weekly (4 weeks), and monthly (1 month) sampling schedule. Under these conditions, sampling of 0.2% of the body weight in blood produced deleterious effects in the fish. By correlating measured pituitary and plasma

gonadotropin levels with spermatogenesis in testes of rainbow trout, Breton and Billard [475] demonstrated that control fish exposed to natural photoperiod and temperature from February to June did not show advancement of spermatogenesis, whereas those held at either 8° or 16°C and exposed to decreasing photoperiod showed complete induction of spermatogenesis, mediated through a rise in gonadotropin secretion. McHugh and Heidinger [476] found that 509-day-old striped bass (*Morone saxatilis*) fry displayed diving, rapid swimming movements, and some mortality when exposed to light shock (1 238 lm after 3-hour dark), whereas 15 to 33-day-old fry had no discernible reaction. Handling stress (10 seconds in dip net) produced some mortality, loss of equilibrium, erratic swimming, or loss of activity during the 1-hour postshock observation period. Curry and Kynard [477] suggested that galvanonarcosis, the state of muscular relaxation produced by constant, direct current, was a potential alternative to chemical anesthetics for long periods of fish immobilization. Individual and group activity decreased in yearling rainbow trout and channel catfish (*Ictalurus punctatus*) subjected to direct current narcosis for 0.5 and 3.0 hours. Except for trout narcotized for 3.0 hours, all fish recovered to levels of activity seen in control fish within 24 hours. A book, edited by Danyulite,[478] summarized data on the survival of fish, crustaceans, and organisms used as food by fish after exposure to pulsating, direct and alternating current, and on the effect electric fields have on the reproduction, early development, and physiological state of the animals.

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